

# THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—20TH YEAR.

SYDNEY, SATURDAY, DECEMBER 16, 1933.

No. 25.

## Table of Contents

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ORIGINAL ARTICLES—	PAGE.	CURRENT COMMENT—	PAGE.
"Tubal Block and Other Adnexal Lesions from the Aspect of Sterility", by S. C. FITZPATRICK, M.B., B.S. . . . .	809	Hypertensive Encephalopathy in Nephritis . . . . .	828
"Lead Poisoning", by G. C. WILLCOCKS, M.B., Ch.M., F.R.C.P. . . . .	813	Artificial Pneumothorax . . . . .	829
"Basophilia and Lead Excretion in Lead Poisoning", by CHARLES BADHAM . . . . .	816	ABSTRACTS FROM CURRENT MEDICAL LITERATURE—	
"The Queen Charlotte's Hospital Report on Puerperal Fever: A Summary with Some Comments", by FRANK A. NYULASY, M.D., Ch.B. . . . .	821	Physiology . . . . .	830
"The Position of the Urinary Bladder", by H. FLECKER, M.B., Ch.M., F.R.C.S. . . . .	823	Biological Chemistry . . . . .	830
REPORTS OF CASES—		SPECIAL ABSTRACT—	
"An Unusual Drug Eruption", by J. M. O'DONNELL, M.B., Ch.M. . . . .	823	Lead Poisoning at Mount Isa, Queensland . . . . .	832
REVIEWS—		BRITISH MEDICAL ASSOCIATION NEWS—	
Treatment by Suggestion . . . . .	824	Scientific . . . . .	833
Dermatology . . . . .	825	Nominations and Elections . . . . .	835
Infra-Red Rays . . . . .	825	CORRESPONDENCE—	
An Experiment in Sex Education . . . . .	825	The Health of the People of Papua . . . . .	835
Neurological Examination . . . . .	826	DIARY FOR THE MONTH . . . . .	836
Neuropathology . . . . .	826	MEDICAL APPOINTMENTS VACANT, ETC. . . . .	836
Rheumatoid Arthritis . . . . .	826	MEDICAL APPOINTMENTS: IMPORTANT NOTICE . . . . .	836
Surgery in Tablet Form . . . . .	826	EDITORIAL NOTICES . . . . .	836
LEADING ARTICLES—			
Lead Poisoning in Industry . . . . .	827		

### TUBAL BLOCK AND OTHER ADNEXAL LESIONS FROM THE ASPECT OF STERILITY.<sup>1</sup>

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STERILITY is a problem of unusual interest and exceptional importance alike to the patient, the practitioner and the State.

To the practitioner the problem is one that often entails a long investigation of many possibilities. But as Forsdike<sup>(1)</sup> says, "to achieve success in a case of sterility confers greater happiness upon the people concerned than any other form of surgical procedure, and the search for a solution should not lightly be given up".

The majority of sterile women cannot in the present state of our knowledge be brought to fer-

tility. There is, however, a definite if small percentage of cases which offer reasonable chances of success if proper investigation and treatment are carried out. I shall limit the scope of my paper to the consideration of typical cases of tubal block and some adnexal lesions which have occurred in my own practice. These will illustrate some of the possibilities, probabilities and disappointments in attacking this problem.

Tubal block may be due to infection, injury, displacement, maldevelopment, new growths. Infection is by far the most important cause. In all cases described the husband was first examined and proved potent and fertile.

#### Case I.

*Pelvic Infection; Sterility; Salpingostomy; Ruptured Ectopic Gestation.*

Mrs. J. J. S., aged thirty-eight years, was married in 1918. She was a nullipara, and developed a genital tract infection, presumably gonorrhoeal, within two weeks of marriage.

<sup>1</sup>Read at a meeting of the Victorian Branch of the British Medical Association on July 22, 1933.

Operation for removal of extensive pelvic adhesions, omental and ileal, and to free ovaries from investing adhesions, was carried out in 1929. The patient was insistent in her request to have every chance given for pregnancy. Tubal inflation proved non-patency. On September 25, 1930, I freed the right tube from adhesions, clearing the fimbriae, resected the left ovary, removed the left tube, and divided other adhesions. She became pregnant the month after leaving hospital, but on January 24, 1931, I regretfully had to do an urgent laparotomy for a right tubal abortion.

This case represents the commonest cause of sterility in women, infection of the pelvic peritoneum. Only those in which the mucosa of the tubes is almost normal and in which the ovaries can be divested of adhesions offer any hope of success by salpingostomy; but as gonorrhoea damages the tubal plicae almost invariably, tubal gestation is more liable to occur.

#### Case II.

*Ectopic Gestation; Intraligamentous Rupture; Sterility; Salpingostomy; Three Pregnancies.*

Mrs. A. L., aged thirty years, married ten years, a nullipara, on May 12, 1925, suffered from rupture of a left tubal pregnancy between the layers of the broad ligament. My *locum tenens* treated her non-surgically. The resultant hæmatocele by 1927 had produced a large tender cyst. On account of her continued sterility I suggested a Rubin's inflation test. This showed non-patency. On August 3, 1927, at operation, a large simple unilocular cyst was removed from the left broad ligament, together with a damaged tube. Fine adhesions turning in the fimbriae at the abdominal ostium of the right tube were gently divided, and a probe was passed without force as far as the interstitial part. Contraception was recommended for twelve months. Conception in April, 1929, resulted in a fine male baby on January 18, 1930, and two other fruitful pregnancies have followed.

This case gave the most favourable conditions for a successful salpingostomy, where the fimbriae are adherent, but in a condition to be conserved, and where the minimum of trauma has to be done so that no raw area is left to reform adhesions. Factors of success are great gentleness of handling, fineness of needles and sutures, complete hæmostasis and peritonealization, proof of regained patency by injecting five cubic centimetres of 0.5% indigo-carmin through the tube till it shows in the vagina, and an "arum lily" type of ostium when resection is necessary. At this date, one would have done a "lipiogram" before operation. This would have shown the clubbed extremity typical of the closed abdominal ostium. Also one would have inflated the tube once or more times following operation to prove and maintain patency.

#### Case III.

*Primary Sterility; Adenomyomata of Tube; Salpingectomy.*

Mrs. F. A., married four years, a nullipara, sought investigation for sterility. Her uterus is under average size and menstrual loss small, lasting two days. Inflation showed non-patency. A lipiogram showed a tubal block in the ampulla of the left tube and an isthmal block of the right tube. At the patient's urgent request I opened the abdomen. Two small tumours in the left tube, three in the right tube and one at the right cornu, all about five millimetres in size, were present, on section proving adenomyomata. I resected the left tube 3.1 centimetres (one and one-quarter inches) from the free end so as to take

away the tumours, formed an ostium and proved patency by injection of indigo-carmin. Inflation was repeated at intervals beginning at the tenth day and a lipiogram proved patency at three weeks. Under-development of the uterus and possibly endocrine deficiency are also present here.

This case is far too recent to put down as a failure, but it would be far too optimistic to expect a success. I have included it as showing one of a class of cases in which the actual cause of block cannot be shown without laparotomy. Fibroids of the tube are rare and are considered by some to be the expression of a mild infection. Nodular salpingitis is a distinct clinical entity from tubal fibroids.

Regarding post-operative use of inflation, I should like to hear the views of others on how soon this should be begun, how often it should be repeated, and for how long it should be persisted with. One wonders how soon intraperitoneal adhesions, due to aseptic clot on a serous membrane lesion, become sufficiently strong to withstand an intratubal pressure of, say, two hundred to three hundred millimetres of mercury. And would the use of iodised oil post-operatively tend to lessen the chance of adhesions at the newly-formed ostium?

#### Case IV.

*Torsion of the Left Tube Associated with Left-sided Caecum and Acute Appendicitis.*

M. K., aged twenty-two years, a nullipara, complained for eight years of steady left-sided abdominal pain, in attacks lasting twelve to seventy-two hours. Vomiting occurred after the onset of the pain. Two attacks in 1933 were more severe. No dysuria or urinary abnormality occurred. X ray investigation excluded urinary calculus and showed a left-sided caecum with tender appendiceal base. Operation revealed a rigid appendix more inflamed than expected, in the left iliac fossa and unexpectedly an ovoid purple tumour (specimen shown) in the pouch of Douglas due to volvulus of the left tube only; the pedicle had one and a half turns in a clockwise direction; it contained blood *débris* only; there was no sign of ectopic pregnancy.

Did a previous twist or appendicitis in this case lead to the sealing of the ostia and the last severe attack cause the hæmosalpinx? Torsion of the uterine adnexa is relatively uncommon, and is unlikely to be diagnosed before laparotomy is done. Coghlan<sup>(2)</sup> of Sydney, however, describes two cases he met with in one day. It is to be noted that normal tubes can twist through 90°, and that all tubes which become abnormal by sealing of the ostia and retort-shaped distension tend to twist as they swing backward into the pouch of Douglas, but extreme twisting is usually prevented by inflammatory adhesions.

From the point of view of sterility, bilateral volvulus of the tubes would mean complete mechanical block and neither the inflation test nor a lipiogram would reveal the cause. The case given is an example of a developmental fault leading to tubal block.

The presence of the caecum in the left iliac fossa, due here to uncommonly advanced developmental torsion of the mid-gut, should make one alert to the possibility of other associated developmental abnormalities.

Hofmann<sup>(3)</sup> describes a torsion of the right tube occurring during the eighth month of pregnancy leading to severe abdominal pain and a tumour in the ileo-caecal region. Laparotomy for suspected appendicitis showed a right tube twisted in its proximal third through 360° with hæmorrhagic infiltration but no hæmatosalpinx. Unusual length and short mesosalpinx were predisposing factors.

#### Case V.

*Sterility; Persistent False Corpus Luteum; Rupture with Severe Intraperitoneal Hæmorrhage; Operative Removal; Pregnancy.*

Mrs. L. J. H., aged twenty-six years, married three years, was a nullipara; she had used no contraception. On February 23, 1932, she suddenly collapsed with the symptoms and signs of a ruptured ectopic gestation; she was five days past her normal menstrual date. Immediate operation revealed a ruptured luteal cyst in the right ovary, with the pelvis full of blood. Resection of the ovary was done. The menses came on three days after operation. She left hospital in March and became pregnant in June, and was delivered in April, 1933.

Dr. E. S. J. King's report was:

"An old luteal cyst into which hæmorrhage has occurred. Why they result in such severe intraperitoneal hæmorrhage is not known, but is probably hormonal. I do not mean this in the usual general vague sense—quite a lot of scientific work has been done on it. On some occasions they have been fatal."

Severe intraperitoneal hæmorrhage as the result of rupture of a *corpus luteum* was described last century under the term of apoplexy of the ovary. Many cases have been reported, but none have been correctly diagnosed before operation. Two clinical types emerge, that simulating acute appendicitis, and the other, fulminating, simulating ruptured ectopic gestation. Both types call for laparotomy. Schickele<sup>(4)</sup> and Penny<sup>(5)</sup> each describe cases in which the hæmorrhage ended fatally.

What is the factor which causes the hæmorrhage into the cavity of the follicle to become excessive? Disease of the ovarian vessels, trauma, some hormonal imbalance have each been suggested.

What is the influence of a persistent *corpus luteum* on fertility? The persistence of a *corpus luteum* in cattle is said to be responsible for many sterilities which are known to be fairly common in herds of pure breed. Expression of *corpus luteum* in cattle by compression of the ovaries *per rectum* in these animals which miss their heat is followed by heat in two to five days. (T. S. Gregory, B.V.Sc., confirms this statement.)

Ochsner<sup>(6)</sup> concludes that expression or excision of false *corpus luteum* in the human female is followed by menstruation in twelve to thirty-six hours, and he gives details of nine cases.

Johnstone<sup>(7)</sup> of Edinburgh considers that while this may be true for cows there is no proof of the same in women, although some cases of sterility with amenorrhœa may be due to it, and presumably to excessive or uninterrupted secretion of "Prolan B", or sterility in such cases may be due to a thick ovarian cortex, or due to dense adhesions preventing the maturation of the follicle and the consequent

death of the ovum in the follicle. Laparotomy to deal with the thick cortex and adhesions should be done.

Kraue states that after removal of the pituitary in rats, follicle ripening ceases and the *corpus luteum* persists for a long time. No degenerative changes were noticed in such *corpora lutea*. The uterine mucosa becomes atrophic. It cannot be assumed from this that the persistent *corpus luteum* contains no hormone. Follicle hormone is essential to prepare the uterine mucosa for the pre-gravid changes induced by the luteal hormone. When the pituitary is removed, both maturation of the follicle and the production of "folliculin" or "œstrin" is decreased, and so no changes occur in the uterine mucosa.

Beckwith Whitehouse<sup>(8)</sup> gives a very clear account of a case in which he excised the *corpus luteum* and produced a period several days later and well before the usual time.

Cannon<sup>(9)</sup> states that animal experiments show that the life of the *corpus luteum* can be definitely prolonged by continuous administration of the beta hormone of the pituitary gland.

#### Case VI.

*Secondary Sterility; Endometrioma of Pouch of Douglas; Operative Removal; Pregnancy.*

Mrs. A. D., aged twenty-seven years, with one child, nine years old, suffered two years of pelvic pain, severe dysmenorrhœa, dyspareunia and diarrhœa. One June 23, 1929, I removed a small puckered mass showing recent bleeding and adhering the sigmoid colon to the back of the uterus. No other areas of endometriosis were seen. Pregnancy followed in October, 1932, and delivery in July, 1933.

Endometriosis is a disease of comparatively young women; the tumours are slow-growing and therefore exert their influence over a considerable period of the menstrual life. Sterility is therefore generally a paramount symptom of intraperitoneal endometriosis, partly owing to the lesion itself which leads to dysmenorrhœa, dyspareunia and functional sterility, and partly from complicating lesions.

My treatment of this patient may be criticized as dangerously conservative. Much more radical treatment is generally advised, for example, hysterectomy, double oophorectomy, or radiation, but in these comparatively young women, ovarian conservation is very desirable. Careful destruction of every visible spot of the abnormal tissues by electro-coagulation or other means gives the advantage of continued menstruation and possibility of subsequent childbirth to offset the risk of recurrence. Conservatism is of course not permissible in the large multiple and widespread lesions.

#### Case VII.

*Sterility Four Years; Tubal Inflation; Pregnancy.*

Mrs. T. T., aged thirty-eight years, married four and three-quarter years, consulted me for sterility. Inflation was carried out and patency was proved at 90 millimetres. She became pregnant in the following month and was confined on October 26, 1931.



Tubal inflation is now accepted as a therapeutic method for opening closed tubes. Rubin, in a total of 2,000 cases of infertility, reported 205 cases of pregnancy following utero-tubal inflation. Forsdike,<sup>(10)</sup> in a series of 100, reported 31% of pregnancies.

What is the pathology of these occlusions which give way before gas pressure? Kinking, "stickiness", spasm, strictures, minute adhesions have each been suggested. Goodall<sup>(11)</sup> considers that the great majority of those that yield are strictures at the intramural part of the tube and are due to a peritubular lymphangitis and cellulitis of a mild nature.

Forsdike does not claim that all of these successful cases are wholly in consequence of inflation. Two other possible factors have to be remembered: (i) the dilatation of the *os uteri* necessarily incurred during instrumentation, and (ii) coincidence.

The commonest surgical measure employed for cases of sterility was formerly dilatation of the cervical canal and it was regarded as the ultimate resort. A small percentage of women became pregnant after the operation and probably in consequence of it. Now the dilatation involved in passing the uterine sound in Rubin's test, though to a much smaller degree, may have an equally fortunate result.

There is, however, another aspect of the apparently simple operation of dilatation of the cervix. Is it not possible that dilatation of the cervix as usually carried out by the use of solid dilators of the Hegar type may lead to clearance of "sticky" or kinked tubes by raising intrauterine and intratubal pressure? I suggest that this may occur in a few cases. I have tried to obtain definite experimental evidence of this.

During the course of dilating the cervix in several women I placed two to three cubic centimetres of iodized oil into the cavity of the uterus as soon as the size of the dilatation permitted. An X ray photograph was taken at this stage and again at three other stages of the dilatation.

The first film shows the iodized oil in the cavity of the uterus only. Subsequent films show the increasing sizes of the solid dilators *in situ* and iodized oil in the lumen of the tubes and finally reaching the peritoneal cavity. Of course, once the oil enters the tubes, peristalsis in the tubes may convey the oil to the peritoneal cavity.

May this not be a possible explanation of the success sometimes following the old operation of dilatation of the cervix for sterility? The uterus acts as the barrel and the solid metal dilator as the piston of a syringe. A pressure of only thirty to forty millimetres of mercury and only four to five cubic centimetres of oil are needed to fill the uterus and tubes.<sup>(12)</sup> Fluid *débris* or air in the uterine cavity might well be the media by which "inflation" of one or both tubes occurs. This fear of danger from solid uterine dilators was at the back of the mind of F. W. Ramsay<sup>(13)</sup> when he designed

a hollow dilator figured in the "Medical Annual" of 1903, page 729.

Tubal inflation has undoubtedly helped to crystallize our knowledge of sterility. It has taught us the futility of a large proportion of the salpingostomies performed before Rubin's discovery.<sup>(14)</sup>

Salpingostomy and other plastic operations on the tubes have so seldom been followed by pregnancy in the past that we have been hardly justified in advising healthy and comfortable pain-free women to undergo abdominal sections for the one complaint of sterility.

As its value is appreciated, inflation will be used more often after pelvic infections have subsided, whether post-abortion, puerperal, gonococcal, and after ruptured ectopic gestations *et cetera*. The history of every new method or operation is that of over-enthusiasm, "exploitation", too wide an extension of the indications, a period in which it falls under disrepute, and then gradual return of the pendulum to a sane mean.

#### Conclusion.

In conclusion I should like to ask what place should the new methods of investigation of tubal block hold as far as the general practitioner is concerned.

Some maintain that tubal inflation as a diagnostic and therapeutic procedure is outside the scope of the general practitioner; that it is a special method of investigation and should be restricted to use by specialists. It is true that there are definite dangers and contraindications and fallacies to avoid; but the dangers and contraindications to the use of inflation and iodized oil are precisely the same as for cervical dilatation for sterility. That is, there should be complete absence of genital tract infection or suppuration, absence of pelvic tenderness and swellings and fever; absence of grave cardiac, renal and lung disease. But surely tubal inflation, with due regard to the contraindications, is a safer method of investigation and treatment and often of more use than dilatation of the cervix.

#### Acknowledgements.

I gratefully acknowledge the help readily given by Professor Marshall Allen and Mr. T. S. Gregory, B.V.Sc.

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### LEAD POISONING.<sup>1</sup>

By G. C. WILLCOCKS, M.B., Ch.M. (Sydney),  
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Sydney.

It would seem that there is little to be said on the subject of lead poisoning that is new at this time. Nevertheless, the condition is prevalent and the prophylaxis and treatment still leave much to be desired.

By Dr. Badham's courtesy I have examined the records of some two hundred men who have suffered from lead poisoning during the last four or five years. I have studied the records of fifty patients admitted to Sydney Hospital for plumbism during the last ten years; and I have to thank Dr. J. V. Duhig, of Brisbane, for permission to read and use the report of the recent commission on the Mount Isa mine in Queensland, May, 1933. From other sources, including my own experience, I have added material.

I ask you to bear with me in the unsatisfactory task of trying to summarize this very important subject, since only a very brief *résumé* is possible in the time at my disposal tonight.

#### Mode of Entry.

Lead poisoning is caused mainly by inhalation and less by ingestion of lead particles, the more soluble lead compounds being much more dangerous. By inhalation the lead goes straight into the circulation, whereas when ingested much of the lead is caught by the liver and excreted in the faeces, the main path of excretion. Absorption through the skin may occur, as in the use of face powder, and the injection of lead in the treatment of cancer is a well known cause of plumbism.

I shall deal mainly with the industrial aspect of lead poisoning and will not discuss the rare and curious modes of poisoning.

#### Occupations.

The occupations in which plumbism occurs most frequently at the present time are those of lead mining, the making of batteries or accumulators, and lead smelting in various trades.

At Mount Isa mines in Queensland over two hundred men have been on compensation for lead poisoning within the last few months. In May, 1933, following the threat of a strike, a commission investigated the conditions there. It was found that lead was being inhaled and ingested by very many of the staff, with serious results. Measures of prevention were recommended which should be effective; among these was a serious attempt to educate the employees, by lectures and notices, of the risks. I show here one of the notices, which might well be widely displayed in factories using lead.

WARNING NOTICE.—Seek medical advice and blood examination if not well.

(1) Always use safety apparatus provided.

(2) Wear your respirator.

(3) Take regularly any medicines or other treatment provided. Medicine and pills for cleaning out your system are available.

(4) Keep your body clean and have your hair cut frequently.

(5) Bathe at the end of each shift; give your hair especially a thorough cleaning and change to clean clothing.

(6) Change and wash working clothes frequently.

(7) Wash hands before eating. Never eat food in a dusty atmosphere. Always eat in crib room.

(8) Rinse the mouth out before eating or drinking.

(9) Never roll cigarettes at work. Make enough beforehand to last the shift. Fill your pipe with clean hands.

(10) Keep body excretions free. Never allow yourself to become constipated. Epsom salts are cheap and effective. Be sure to have a motion every day.

(11) Eat a substantial meal before coming to work.

(12) Eat plenty of nutritious food.

(13) Avoid all excess, particularly alcoholic beverages.

Other measures for laying the dust by wetting and for removing it by fans were also advised.

In Sydney, workmen in accumulator factories have provided the bulk of the cases of plumbism of late years. The work consists in smelting lead, making lead paste, applying it to battery plates, baking the plates in an oven, dismantling batteries, and so on. The smelting and baking give rise to fumes or vapour of lead, which probably cause the plumbism very quickly; that is to say, in from one to sixteen weeks.

In all occupations in which lead is used, the susceptibility of the individual to lead is of the utmost significance; some men are rapidly poisoned, others have great tolerance. Young men and men unaccustomed to lead work are especially prone to plumbism within a few weeks of starting such work.

Details of all the one hundred and fifty trades in which plumbism may occur may be found in text books; suffice it to say that it very often occurs in occupations where a lead hazard is not at first suspected, and it is comparatively rare in some occupations, such as plumbing, soldering and linotyping, in which it might be expected to occur more often.

Dr. Badham has prepared a chart showing the occupations of five hundred persons examined for plumbism during the last few years (see accompanying table).

<sup>1</sup> Read at a meeting of the New South Wales Branch of the British Medical Association on October 26, 1933.

Table showing the Occupation of 515 Individuals Investigated for Lead Poisoning by the New South Wales Division of Industrial Hygiene in the Past Seven Years (1927-1933) and Their Diagnosis.

Industry.	Occupation.	Number Examined.	Lead Poisoned with Disability.	Not Lead Poisoned or Lead Poisoned without Disability.
Printing	Hand composing	22	2	20
	Machine composing	11	—	11
	Other processes	21	1	20
Manufacture of electric accumulators	Pasting	41	35	6
	Mixing	28	23	—
	Assembling	15	11	4
	Burning	12	7	5
	Moulding	18	8	10
	Formation	16	6	10
	Handling dry plates	52	47	5
	Repair work	8	2	6
	Lead oxide plant	5	1	4
	Cleaner	3	2	1
Painting	Coach painters	17	7	10
	House painters	70	15	55
	Spray painters	22	—	22
	Bridge painters	10	6	4
	Commercial painters	5	2	3
	Ship painters	8	—	8
	Paint factory	1	—	1
	Lead corroding	5	4	1
Plumbing and soldering		27	—	27
Glass works		6	6	—
Vitreous enamelling	Fusers	3	1	2
	Shot blasting old baths	1	1	—
Shipbreaking		1	1	—
Petrol distribution		1	1	—
Smelting of metals	Pump maintenance	18	14	4
Carpenter		1	1	—
Leadlight	Glazer	2	—	2
Lead caulking (pipes)		2	—	2
Brass foundry		5	—	5
Sheet metal workers		9	1	8
Rubber	Hose curing	9	—	9
Galvanizing wire		1	—	1
Pottery works	Kiln hand	1	1	—
	Glazer	1	—	1
Signwriter		1	—	1
Ink maker		1	—	1
Mining		1	—	1
Manufacture of arsenate of lead	Lead miners	5	2	3
Miscellaneous		1	1	—
	Adventitious exposure	1	—	1
	Non-lead worker	17	—	17
Totals		515	220	295

#### Children.

In Queensland and South Australia lead poisoning has been reported quite frequently in children.

In the last ten years only four children in whom a diagnosis of plumbism was made have been treated as in-patients at the Royal Alexandra Hospital for Children in Sydney, and in two of these the diagnosis was doubtful. It is useless to try to draw any conclusions from these data, except that plumbism appears to be exceedingly rare among children in Sydney.

#### Pathology.

Lead is stored in the body, mainly in the bones (95%), and to a less extent in the liver, kidneys and other organs.

The occurrence of intestinal colic and of abortion indicate its effect on smooth muscle (increased tone and decreased motility).

Interstitial neuritis may occur, and the anterior horn cells of the spinal cord may degenerate. Meningeal congestion with increase in spinal fluid, and in the cells of the spinal fluid, has been described. Dr. Badham has records of one case in which a large ulcer was noted over the frontal lobes of the brain. Optic neuritis has been reported

often. Aub, however, is very conservative in his statements concerning the morbid anatomy.

Gastric ulcer is said to be common in lead workers. I have noted three such among the records of three hundred cases of plumbism.

Congestion of the kidneys occurs, and acute nephritis; apparently the acute condition may merge into chronic nephritis, but Aub and his co-workers doubt this. Chronic nephritis might reasonably be expected when the frequency of albuminuria and granular casts is known.

However, I could find evidence of plumbism in only two out of one hundred consecutive patients with chronic nephritis at Sydney Hospital. There were more clerks than lead workers in this series. Among fifty cases of plumbism at Sydney Hospital there were only two cases of nephritis, one acute and one chronic.

Arteriosclerosis also is such a common disorder that an undue frequency among lead workers is not so obvious now as the text books would lead us to believe.

Aub, after an exhaustive review of the literature, did not include chronic nephritis, arteriosclerosis, gastric ulcer and other degenerative conditions among the sequelæ of lead poisoning. He records that "some say that" the above conditions are more

frequent in lead workers; and yet in another place he states "that the late manifestations of lead poisoning are largely degenerative, and are caused by the continued action of the lead stream, shown strikingly by the frequent appearance of arteriosclerosis, contracted kidneys, high blood pressure and hypertrophy of the heart".

#### Symptoms.

Among the patients reviewed, abdominal pain (lead colic) was the most frequent symptom. It is due to increased tone and decreased motility of the smooth muscle of the bowel. Unlike ordinary intestinal colic, the pain tends to persist for hours or days; hence the reason for the not infrequent operation, for appendicitis, in plumbism.

The pain may be in the upper or lower portion of the abdomen; it may be mild or severe. Anorexia, nausea and vomiting are very frequent symptoms. Constipation is rarely absent when colic occurs, but it is not constant at other times. The lack of appetite, the furred tongue and other symptoms indicate the severe effect on the gastro-intestinal tract.

#### Nervous Symptoms.

Headache is often complained of, and convulsions occurred four times in three hundred cases reviewed. Aub considers that these symptoms are due to productive meningitis rather than to encephalitis. Insanity was noted twice in this series, but its relationship to lead was not proven.

Weakness of one or both arms, weakness of the legs and general weakness were among the most common complaints. Wrist- and foot-drop were rarely noted, and in this series, when such signs occurred, a varying degree of alcoholic indulgence was usually recorded. A similar history was obtained in two of the four patients who had convulsions.

Pains in the limbs and joint pains were frequent. Exhaustion and tiredness were the foremost symptoms in several severely poisoned patients.

Definite paresis of muscles, especially of the forearm muscles, may be rarely found. The reflexes, knee, ankle and arm jerks, were affected three times only.

#### Signs.

Pallor is so definite in some patients as to be pathognomonic. A blue line on the gums occurs in over 60% of those whose plumbism is diagnosed. Albuminuria and renal casts were present in nearly every patient at some time. Jaundice was observed three times, probably due to hæmolysis (an indirect positive Van den Bergh reaction was obtained).

Basophile degeneration of the red cells is the most valuable sign; it is very rarely absent, but I have seen four patients, in whom the symptoms were probably due to lead, in whom this sign was absent. (Dr. Badham will deal with this aspect.)

Anæmia of all degrees occurs, diminution of hæmoglobin and in the number of red cells especi-

ally, but poikilocytosis, polychromatophilia, changes in size, and rarely nucleated red cells may be observed.

On the subject of basophilia, Aub states that a punctate basophilic degeneration, best demonstrated by dilute alkaline methylene blue stain, is the most characteristic change in the blood. This appears during absorption and may continue for many months after absorption of lead has ceased. These cells are probably degenerating young reticulated cells. A few may be found in cases of pernicious anæmia, leuchæmia, and sometimes even in normal blood; but such large numbers as one hundred per million red cells do not appear in adults in any disease except lead intoxication. Stippling is therefore one of the reliable criteria for establishing the diagnosis of lead absorption.

#### Diagnosis.

Diagnosis is made on the occupation and on the presence of two or more characteristic symptoms or signs. Severe lead colic is fairly characteristic, as is the true blue line (bismuth injections for syphilis may also cause this). Pallor is a valuable sign.

The symptoms and the appearance of the individual may be quite enough, with the changes in the blood, to establish the reality of poisoning; but Dr. Badham has had more than one patient in whom the basophilia was high—5,000 per million red cells—and yet the patient stated that he felt perfectly well. So that basophilia does not always indicate incapacity.

Alcoholism definitely aggravates the symptoms. Syphilis and other anæmias, such as pernicious anæmia, have sometimes caused a doubt in the diagnosis. Each case must be considered individually after taking a careful detailed history.

#### Prognosis.

Away from lead work recovery takes place as a rule in from one to sixteen months. Recovery from neuritis is slow. I have known albuminuria to persist for years, but the early history of this patient was uncertain. Undoubtedly lack of proper food, indulgence in alcohol and lack of medical supervision may cause an undue prolongation of the symptoms.

Some of the men develop a fear of the complaint and do not sufficiently try to reestablish their health by suitable fresh air, exercise and other measures. There is a tendency for a compensation neurosis to develop.

#### Prophylaxis.

There is room for improvement in the supervision of occupations in which lead is used. Perhaps the onus is now on the insurance companies to maintain adequate and expert medical supervision of the dangerous trades.

It is certain that a great deal too much serious ill health is caused to workmen, and the community is put to great expense in paying compensation to these unfortunate individuals at the present time.



It is a very haphazard state of affairs when twenty or thirty men have to be poisoned before adequate precautions are taken. Unfortunately the men do not always take reasonable care of themselves.

Cleanliness of hands and face, the use of rubber gloves, the use of proper respirators in dusty trades, adequate ventilation, and exhaust fans to remove the dust or vapour are well known methods of prevention. I see no reason why vacuum cleaners should not be used to remove lead dust also.

Because a high calcium diet favours storage of lead, a glass of milk at breakfast and between meals is advisable. A daily dose of salts, by keeping the bowels free, will help to prevent absorption from the bowel.

#### Treatment.

The patient must be removed from the occupation in which he has contracted plumbism, until his symptoms have subsided, his signs have disappeared, and his blood is normal, or nearly so. This may take from one to eighteen months.

Such a man is known to be susceptible to lead (for some are not susceptible), and greater precautions should be taken if he resumes his previous occupation. This is not always done, and the man is often off work again with the same symptoms in two or three weeks. Obviously he should not resume the same occupation unless more adequate protection is enforced.

Lead is stored in the calcareous part of the bones as the insoluble tertiary lead phosphate. There is a close analogy between the storage and excretion of lead and calcium, so that when acute symptoms occur the administration of calcium lactate (one gramme twice a day) and large quantities of milk (one quart a day) are indicated to promote storage of lead which is in circulation. Aub and Donald Hunter both state that a high calcium diet promotes storage.

Lead colic and encephalopathy are urgent indications for this form of treatment. Calcium has an antispasmodic effect on involuntary muscle, and a high calcium diet usually relieves colic in forty-eight hours.

In severe cases the slow intravenous injection of fifteen cubic centimetres of a 5% solution of calcium chloride relieves pain at once; it may be repeated after two hours. Incidentally Aub states that he has relieved renal and gall-stone colic in this way.

When the acute symptoms have subsided, an attempt may be made to promote the excretion of lead by reducing the alkali reserve; an acidosis promotes the excretion of lead and calcium. This procedure must be closely watched, since acute symptoms, such as nausea, headache, colic and even convulsions, may occur if too great an amount of lead is liberated from the bones into the circulation.

Many methods of promoting the excretion of lead have been investigated in recent years. The old-fashioned mixture of magnesium sulphate and potassium iodide, in small doses (0.12 to 0.42

gramme or two to seven grains), is known to promote excretion. Dilute phosphoric acid and acid sodium phosphate are effective. The most satisfactory method is a diet containing only one hundred milligrammes of calcium, and to give one gramme of ammonium chloride in water six times a day for two or three weeks. If toxic symptoms occur this régime should be suspended and a high calcium, high milk diet given to promote storage.

Donald Hunter records that the use of parathyroid extract (parathormone) caused excretion of lead in far greater amounts than potassium iodide, ammonium chloride or phosphoric acid, but owing to its effect in throwing so much lead into the circulation there was a danger of acute toxic effects of plumbism being caused.

#### BASOPHILIA AND LEAD EXCRETION IN LEAD POISONING.<sup>1</sup>

By CHARLES BADHAM,

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THE industrial hygienist who is concerned with a particular field of medicine finds that, of the industrial poisonings, lead is easily first, and, moreover, that it occurs in numerous industries and that its presence has just as often to be disproved as to be proved. To him any method of examination which will enable an accurate diagnosis of lead poisoning is a very real benefit.

I have, during the past ten years, watched with anxiety and relief the development of the methods practised by my Division for the diagnosis and prevention of lead poisoning. The basis of these methods has been the study of the punctate basophilia or stippling of the red cells and the urinary excretion of lead.

Before I proceed to detail certain points in these two problems in lead poisoning, I wish to convey my thanks and to place on record the debt I owe to the untiring zeal and efficiency of my colleagues, Dr. H. B. Taylor and Mr. H. E. G. Rayner, for without their aid and interest my Division could not have done the work it has done. I am satisfied that these methods have led to a very great improvement in accurate diagnosis and the prevention of the gravely incapacitating lead palsy. Ten years ago the typical picture of a case of lead poisoning was that of a painter with a dry belly-ache and (happily for a diagnosis) a blue line; the typical blood picture was unknown or not accepted; a crude, tedious and inaccurate qualitative examination of the urine ended in a report that lead was present or absent.

A sophistry called lead absorption separated the sheep from the goats, the mild anæmias from the severe colics and palsies. The man accepted as suf-

<sup>1</sup> Read at a meeting of the New South Wales Branch of the British Medical Association on October 26, 1933.

fering from lead poisoning was satisfied that he was suffering from a very grave, probably incurable disease, and cases of chronic neurosis as sequelæ were common. The milder sufferers—those with so-called lead absorption—were left at their work to mature till they complained of severe colic, anæmia and palsy. Today, with attention to the blood picture, men are generally removed from lead work before these signs and symptoms appear or a fatal encephalopathy is produced. When they do occur, it is generally in a new lead process conducted by persons ignorant of the inherent dangers. It was difficult to convince some of my colleagues that our standards of diagnosis were worth while, especially one who was in Broken Hill in the carbonate days and who "knew lead poisoning when it was lead poisoning"—when, in the words of Dr. Bartley in 1891: "That at the lead mines at Broken Hill the miners are very susceptible to lead poisoning, often in a very severe form, more generally colic, but not infrequently coma and epileptiform convulsions." Eventually, however, this colleague came to see that it was more economical for the Government Insurance Office to accept our advice and to remove men with mild lead poisoning from lead work until the blood was restored to a normal state, than to carry severe cases over long periods.

The appeal of this work should not be only to those who practise in industrial areas, for the acceptance of the fact that the simplest and most delicate test for lead poisoning lies in a competent examination of the blood for stippled red cells, will enable a diagnosis of obscure anæmias caused by a leaded water supply.

#### Nature of Lead Poisoning.

Lead poisoning is essentially lead anæmia (that is, if we except certain very acute and rare conditions). This anæmia is caused by the destruction of red cells by lead circulating in the blood stream, and it is evidenced by the reduction of the number of red cells and of the amount of hæmoglobin of the blood. The action of lead is to cause the red cell to become fragile and to undergo hæmolysis. This hæmolysis causes hæmatoporphyrin to appear in the urine, and its presence has been used on the Continent as a test for lead poisoning. This lead anæmia is not found in anyone poisoned by lead without the occurrence of stippled red cells. With improvement in the percentage of hæmoglobin and the number of red cells, the punctate basophilia is reduced and will disappear long before a lead palsy, but not before a lead colic; but so long as there is sufficient lead in the circulating blood to cause a lead anæmia there is basophilia.

#### The Nature of the Basophilic Granules.

In regard to the nature of the granules seen in the red blood cells with punctate basophilia, Davidson<sup>(1)</sup> concludes that:

The three basophilic conditions, polychromasia, punctate basophilia and reticulation, are direct evidence of bone marrow activity. While all three phenomena are closely

allied, since they represent different aspects of the same substance, i.e., the basophil material of young erythrocytes, punctate basophilia is an aspect seen only in pathological states; it is an indication of pathological alteration as well as mere youth.

He also concludes that:

Reticulocytes are in no way characteristic of any individual pathological condition, but are merely evidence of blood regeneration.

Whitby and Britton<sup>(2)</sup> summarize their observations as follows:

(1) Polychromasia and stippling are both manifestations of the phenomenon of reticulation. (2) The polychromatic cells of Leishman-stained films and the reticulocytes of supravital stained films are identical and normal. (3) Stippling is the same chromatic substance slightly altered by lead or other poison. The alteration appears to have no effect on the efficiency or life of the cell.

To me it appears that this pathological alteration of the basophilic substance of the immature erythrocyte known as punctate basophilia may be regarded as an index of the red cell destruction by lead poisoning which results in blood regeneration; and just as the presence of reticulocytes shows blood regeneration in various anæmias, so the punctate basophilia of lead poisoning is not only a sign of regeneration in this anæmia, but an index of the amount of red cell destruction, as well as a sign of the continued action of lead.

To quote Davidson:

Stippled cells are never seen in normal blood and, therefore, they are an expression of toxic damage to the blood forming tissues.

In another place he says:

In several cases of severe lead poisoning I have found that when a count of the stippled cells in a preparation stained by Leishman's stain is compared with a reticulocyte count of a vitally stained film from the same blood, a close numerical relation exists between the two types of cell.

Keeping in mind the work of Brookfield<sup>(3)</sup> and Brückner,<sup>(4)</sup> that the sign of punctate basophilia might depend on the reagents used, we have, during the past seven years, been careful to use the same technique in fixing and staining blood slides so that our standard has been constant. We consider stippling as a sign that lead is in the blood stream and that immature erythrocytes have been acted on, and that with particular methods of staining and fixing blood films basophilic granular erythrocytes are revealed in numbers bearing some relation to the amount of lead in the blood. We consider that this stippling of red cells is a definite sign of lead poisoning.

We have not attempted to apply the dark ground method of Nelson, Lockwood and Mackay,<sup>(5)</sup> despite the fact that such a method has been approved by Davidson, for we regard this as one of many variations which will be devised for the enumeration of punctate basophilia, and each method must be subjected to a long series of observations before adoption.

#### Punctate Basophilia in Conditions other than Lead Poisoning.

It has often been stated that punctate basophilia is found in the blood of normal persons and in the

blood of those exposed to various substances, such as arsenic, benzol and cement or in the blood of those who have eaten some particular food. Our experience has been that punctate basophilia is not found in the blood of normal persons in Sydney; and while it may occur in limited degree in secondary and primary anæmias, it rarely causes any difficulty in diagnosis in these conditions.

I believe that the punctate basophilia of the red cells found in so-called normal persons elsewhere comes from the ingestion of lead. If we were to find it even after exposure to such a definite anæmia-producing substance as benzol, we should assume the presence of lead until an examination of the urine showed no increased lead excretion.

If punctate basophilia of the red cells is found in a worker in this State, in the absence of signs of a primary anæmia or of a good cause for a secondary anæmia (or even in their presence if the number of basophile cells is large) one is entitled to assume that it has arisen from a lead intake, and to proceed to investigate the industrial conditions and the lead excretion of the patient.

I refer to industrial workers and not to children and infants. In adults it is easier to examine for evidence of a lead intake either by a knowledge of the working conditions of the patient or by finding the urinary excretion of lead; in children it is more difficult to determine definitely, and I have no personal experience of lead poisoning in children.

Recently Brown,<sup>(6)</sup> Mayers,<sup>(7)</sup> and Kogan and Smirnowa<sup>(8)</sup> have failed to find punctate basophilia of the red cells of normal persons. Let us consider their technique.

Brown used Wright's stain, but the fixative is not mentioned. Mayers used a special eosin methylene-blue and fixed in methyl-alcohol. Kogan and Smirnowa used methylene-blue and methyl-alcohol.

I would quote the last workers:

Although we do not exaggerate the importance of basophilic granules or consider their presence as absolutely specific for lead poisoning, we do believe, however, that we must not underestimate their diagnostic value as a sign of lead poisoning. On the basis of our studies we consider it possible to introduce the term "practical specificity" of the presence of basophilic granular erythrocytes in the blood of workmen in the lead industries.

And in regard to the practical specificity of basophilia as a sign of lead poisoning let me quote Davidson and his colleagues,<sup>(9)</sup> of the Department of Medicine and Public Health, Aberdeen, in their report on an interesting investigation on lead poisoning from water supplies. They say:

Moreover the absence of punctate basophilia from hundreds of blood films stained in this way [they used Lishman's stain] from cases attending the blood clinic in Aberdeen, served as a control in regard to the rarity of punctate basophilia, except where the degree of anæmia was severe.

#### General Remarks.

High basophilia counts are associated with those lead processes in which there is the most marked exposure to lead dust. High basophilia counts are

also seen where there is severe anæmia due to lead; they may occur when there is high lead excretion, but there is often no correlation between the amount of lead excreted by the urine and the degree of anæmia and basophilia.

With a severe lead anæmia a high basophilia count is to be expected.

In regard to the size of the granules of basophile cells, they have been classified as being fine, medium or large granules; and Lane's<sup>(10)</sup> statement that the presence of large granules is indicative of excessive and rapid absorption of lead is usually correct, but there are many exceptions.

Polychromasia is generally present with high basophilia counts, especially those over several thousand. Nucleated red cells are seen only occasionally. Megaloblasts are rare.

#### Lead Excretion and Determination of Lead in Urine.

The lead excretion of normal persons was determined by Taylor and published in 1927 in a report in which I collaborated with him;<sup>(11)</sup> his average figure was 0.02 milligramme per litre for Sydney inhabitants. Kehoe and Thamann<sup>(12)</sup> found a much higher figure of 0.08 milligramme per litre in students. Francis, working for the English Departmental Committee on Tetra-Ethyl Lead, 1930, found that the average for normal London inhabitants was 0.049 milligramme per litre, and of country people 0.023 milligramme per litre. Recently Weyrauch and St. Litzner,<sup>(13)</sup> dealing with German people, stated that the amount of lead they found compares with that established by Badham and Taylor in Sydney, and is a little lower than the normal lead value found by Fretwurst and Hertz in Hamburg.

In lead poisoning one expects to find between 0.10 and 0.30 milligramme per litre, but occasionally the lead excretion is quite low and may not exceed the normal figure. This failure to excrete lead is most marked in cases of severe anæmia. The excretion of large amounts up to 1.0 milligramme per litre is sometimes seen when there has been a great intake of lead. If such a large amount is found on more than one occasion, lead poisoning can be forecasted if not already present.

The figures for lead in faeces are not of the same interest as those for lead in urine, for much of the lead does not reach the general circulation, being absorbed by the liver and passed into the gut.

During the past ten years the method of chemical estimation of lead devised by Taylor has been used by him on more than 1,200 specimens of urine. This method, which has been described by him elsewhere,<sup>(14)</sup> is, in my opinion, worthy of general adoption on account of its accuracy, simplicity of manipulation and one's ability to make a number of examinations, for six specimens can be done in one day. Briefly, the method consists of the separation of the lead from the urine by adsorption on calcium oxalate, the elimination of the adsorbing material, and the determination by means of the very sensitive reaction which takes place between lead salts and sodium bisulphite.



In Taylor's method of lead determination a litre of urine is collected, preferably directly, in a Winchester quart bottle, in which have been placed two cubic centimetres of chloroform. This is to prevent fermentation, which is undesirable.

To the litre so collected are added acetic acid, calcium chloride and a saturated solution of ammonium oxalate. The precipitate of calcium oxalate on which the lead is adsorbed is allowed to settle and is separated from the urine by decantation. This residue is heated on a water bath, filtered and washed. It is then transferred to a platinum dish, evaporated to dryness and heated to convert the calcium oxalate to carbonate. Hydrochloric acid is then added to convert the carbonate to chloride, which is evaporated to dryness, then taken up in water acidified with hydrochloric acid, and then made alkaline to methyl red and filtered. The residue is then dissolved in dilute hydrochloric acid and the lead is determined turbidometrically by the addition of a solution of sodium bisulphite.

#### Diagnosis of Lead Poisoning and the Critical Period for Examination.

From the point of view of medical science, lead poisoning is present in an individual when any pathological change can be demonstrated; when, therefore, the abnormal presence of stippled red cells shows that the young cells produced by the reticulo-endothelial system have been acted on by lead, lead poisoning is present.

Lead poisoning with disability, which is a compensable condition, occurs when the poisoning has produced disabling symptoms or when a definite anaemia is present. It is essential in dealing with lead poisoning with disability that the preventive aspect should always be kept in mind, and no medical man dealing with this industrial disease should demand a high degree of disability. Complaint of abdominal pain, constipation, headache, malaise and joint pains, associated with quite a small degree of anaemia, should be sufficient reason to take a man away from his lead work and to keep him away until his blood is restored to a normal state, by which time there should be no complaint of ill health. In the very mild cases, if work away from lead, no heavier than he has been doing, can be found for such a man, so much the better; but I have always held that before a man who has had lead anaemia should be required to do work heavier than he has been used to, his blood condition should be restored to normal. The critical period for the diagnosis of lead poisoning is when lead anaemia is present. Owing to the medico-legal interest in lead poisoning, one is frequently asked to make a diagnosis in a suspected case when the critical period for making a diagnosis has passed or is passing. It is in these conditions that a lingering basophilia, even if of small amount, becomes of significance if the patient has not been competently examined previously. I am not referring here to the sequelae of lead poisoning.

#### Method of Staining and Estimating Punctate Basophilia.

To find and enumerate punctate basophilia amounting only to a few hundred cells per million red cells, it is necessary for the microscopist to focus his attention on the examination of 50 fields of 400 red cells, and his work will be in vain if the chemical reagents used in fixing and staining the slide are impure or the slide is over-stained. The method employed in my Division during the past seven years is to fix a well made blood film for fifteen minutes in a mixture of equal parts of methyl-alcohol and ether. It is essential that these chemicals should be pure. In the presence of acetone in the methyl-alcohol and certain impurities in the ether, basophilia cannot be demonstrated. For staining we use a 0.7% solution of eosin-azur in methyl-alcohol, which is diluted one part to nine parts of distilled water just previous to staining. Staining is completed in fifteen minutes, when the film should show a faint pink colour. Over-staining should be avoided.

The film is examined with a magnification of 630. In the instrument used under these conditions an average field in an evenly distributed film contains 416 red cells and has 23 cells along a diameter of the field, that is, if the number of cells along a diameter is known, say  $X$ , the number in the field is  $\pi \frac{X^2}{4}$ .

For routine working it is easy to construct a table showing the number of fields examined, the number of cells along a diameter, the number of basophile cells found, and the total number per million red cells.

When a greater degree of accuracy is desired there are several mechanical devices which can be used. We consider that the error in our method of counting is not greater than 20% if a sufficiently large number of fields is examined. A numerical estimation of the stippled cells is more satisfactory than a report that an occasional basophile cell is present or that basophilia is marked. The examination of a number of fields compensates for an uneven distribution of stippled cells, and for this reason such a method of counting is preferable to an absolute count made on a smaller number of fields.

A number of stains are more or less satisfactory for demonstrating punctate basophilia, but we prefer Giemsa staining to Jenner or Leishman stains. We do not favour Sellar's methylene-blue, which gives a poor differentiation and does not demonstrate polychromasia satisfactorily.

#### Standards of Lead Poisoning Adopted by Physicians of Great Works in England and America.

Let us consider now certain standards of lead poisoning recently put forward by the physicians of great accumulator works in England and of a factory engaged in making tetra-ethyl lead in America.

Lane,<sup>(10)</sup> of the Chloride accumulator works, states:

**Diagnosis.**—Great care is necessary here. The mere presence of punctate basophilia is not enough on which to diagnose lead poisoning; it is merely, as has been demonstrated, an indication, if present in a marked degree, of recent absorption of lead. The actual count in different individuals varies; and the time that has elapsed since the last exposure is of great importance. In fact, the longer a worker has been away from his work, the less the value of the count; six months after exposure it is, in most cases, practically valueless. The complete absence of punctate basophilia in three samples taken within a week of exposure and while acute symptoms are complained of, is strong but not absolutely conclusive evidence against a diagnosis of plumbism. The presence of a marked punctate basophilia with large granules, together with such other changes in the blood picture as a fall in hemoglobin, changes in shape and size of red cells, and increase in number of polychromes, is strong confirmatory evidence for a diagnosis of plumbism. Beyond this one cannot go. Small granule counts up to 3,000 are of little or no significance in a lead worker; they merely indicate that he is a lead worker. In making a diagnosis of lead poisoning, great stress must be laid upon the symptoms and the clinical picture, which should be considered in conjunction with the complete blood picture.

My criticism of these standards is that I regard the presence of punctate basophilia as evidence of lead poisoning, even if there is no disability. The complete absence of punctate basophilia I regard as conclusive evidence against a diagnosis of lead poisoning, but, of course, the sequelæ of lead poisoning may be present. The blood picture given above of a worker with a lead intake is not only strong confirmatory evidence for a diagnosis of plumbism, but is, in my opinion, definitely diagnostic.

Lane, in the final report of the Departmental Committee on Ethyl Petrol, gives figures for men who were still working at lead processes in an accumulator works; these show diminished hemoglobin, diminished red cells (one count as low as three and a half million) and basophile cells ranging to 18,000 per cubic millimetre. If I found men with similar blood pictures in the Australian branch of his works, I should have no hesitation in putting them off work as definitely lead poisoned.

Gehrmann,<sup>(15)</sup> who is a physician in charge of a large American plant engaged in making tetraethyl lead, writes:

Each examination consists of a careful search for subjective and objective signs of lead absorption, including a complete blood count, with a careful search for stipple cells. In our stipple cell counts we examine 50 oil immersion fields, and consider the presence of these cells as indicative of lead absorption, but regardless of the number, in no way indicative of the degree of absorption. The degree or severity of lead absorption must be decided by the presence of subjective or objective symptoms and signs, either one or both, together with blood changes as determined by a reduction in the hemoglobin, a reduction in the number of red cells and the presence of any number of stipple cells.

The periodic lead examination must be conducted on a regular schedule and in the event of any doubt or suspicion as to the presence of even very slight signs or symptoms, the patient should be immediately subjected to daily observations until his condition is definitely determined. It is most difficult to set up any definite standard for determining when a man has absorbed sufficient lead to warrant his removal from the work, in order to prevent the appearance of symptoms. In general, I believe it safe to say that when a worker begins to show even a slight digestive change, has lost a little weight, lacks a noticeable degree of his original stamina,

and shows stipple cells with some reduction in the number of the red cells and hemoglobin, he should be removed. To some this might seem like the actual presence of lead poisoning rather than lead absorption, but regardless of the degree, this type of worker can be changed to a lead-free occupation without any loss of time and in a relatively short period be entirely symptom free. In other words, he has not been poisoned to the point of invalidism, has not suffered any lasting effects and can be eventually returned to his original work. The occurrence of absorption in sufficient degree to warrant removal indicates faulty operative methods or failure to follow working rules, and calls for immediate investigation to determine the real source of the trouble and the application of proper remedies.

My criticism of this is that while the factory surgeon will not lightly give up the use of the term "lead absorption", "when he [the worker] has not been poisoned to the point of invalidism", the use of the terms "lead poisoning with or without disability" will appeal to those who make a conscience of what they do.

It is surely against our present knowledge to believe that the stippled cell count is not indicative of the degree or of the severity of lead absorption. If this were so, why bother to make these counts?

#### Prevention of Lead Poisoning.

The obvious duty of an industrial hygienist is the detection of lead poisoning and its prevention.

It should be practicable to conduct lead processes without causing lead poisoning, but at present this ultimate boon is not in sight.

We have urged on the industry which causes the most cases the regular medical examination of the men employed, and conducted routine examination of blood slides of employees. This work, which has entailed the examination of some 1,200 blood smears in the past few years, has aided in the campaign of prevention. It has, moreover, allowed us to forecast trouble and to urge reforms, for the amount of basophilia is in direct proportion to the dustiness of the process. This practice is, in my opinion, preferable to a routine official inspection of these factories.

#### Summary.

In conclusion, I would summarize my opinions as follows:

1. Lead poisoning is present when the first pathological sign of the action of lead can be detected, and this is the presence of stippled red cells.
2. Lead poisoning with disability is present when there is anæmia in mild or severe degree, evidenced by reduction of red cells and hæmoglobin and the presence of stippled red cells with or without symptoms of lead poisoning, as malaise, constipation, joint pains, abdominal pains.
3. If the patient is examined in the critical period for making a diagnosis of lead poisoning, that is, when lead anæmia is present, punctate basophilia is always found, and this sign is practically specific for lead poisoning.
4. Before making a diagnosis of lead poisoning evidence of a lead intake should be sought, and this is to be found in a knowledge of industrial processes or in the amount of lead in the urine, or from the presence of a blue line.

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# THE QUEEN CHARLOTTE'S HOSPITAL REPORT ON PUERPERAL FEVER: A SUMMARY WITH SOME COMMENTS.

By FRANK A. NYULASY, M.D., Ch.B. (Melbourne),  
Melbourne.

A REPORT on 533 cases of puerperal fever received at the Isolation Block of Queen Charlotte's Hospital, London, issued on behalf of the clinical and laboratory staff of the hospital, will be found in the July, 1933, issue of *The Proceedings of the Royal Society of Medicine*. As the subject is of vast importance, and since the report covers twenty pages of closely printed matter, a summary of some of the leading facts should be of general interest. The deaths from sepsis are shown in the accompanying table.

Group.	Number of Cases.	Deaths.	Rate per Thousand.
Booked cases, in-patients .. ..	5,394	4	0.74
Booked cases, out-patients .. ..	4,129	3	0.72
Emergency cases, in-patients .. ..	408	6	14.71
Totals .. .. .	9,931	13	1.3

The mortality from sepsis for all Queen Charlotte's Hospital deliveries was thus 1.3%, whereas the average for England and Wales was about 2%.

Factors bearing on ætiology were on the whole negative, but a few interesting points were observed. Two hundred and eighty-nine patients with puerperal fever were admitted with a history of normal spontaneous delivery (the last three words are in italics), in many cases no vaginal examinations at all having been made. Of these 252 were delivered at term and 28 died, giving a mortality of 11.1%. So that nearly 50% of the infections followed a normal delivery. Further, only 28 of all the patients who died (these patients who died include 61 among 320 patients admitted from outside sources) are recorded as having perineal or vaginal lacerations. The authors state that:

These facts emphasize that some factors other than repeated examinations and lacerations of the genital tract must be sought for as the explanation of a large number of these infections.

Three-fifths of the patients were *primiparæ*. The first two confinements were the most dangerous. Albuminuria was found in 47 cases and definite septic foci in 18.

Intrauterine manipulations were recorded in 202 cases—forceps, embryotomy, extended breech, internal version, rotation and Cæsarean section, besides 44 cases of manual removal of the placenta with six deaths. Lacerations were noted in two-thirds (334) of the patients admitted and in 28 of the fatal cases, so that the mortality of cases in which lacerations are recorded is 8.4%.

*Ante partum* hæmorrhage occurred in 5.2% and *post partum* hæmorrhage in 18% of the patients admitted.

There were 308 cases of local sepsis, that is, of the uterus, vagina or perineum, with no deaths.

There were 81 cases of blood infection in the whole series of 533 cases, and the bacteriology and mortality in the 81 cases were:

- Streptococcus pyogenes* with a death rate of 87.8%.
- Anaerobic streptococci with a death rate of 42.0%.
- Bacillus coli* with a death rate of 16.6%.
- Staphylococcus aureus* with a death rate of 100.0%.
- Other organisms with a death rate of 20.0%.

The total mortality in the 81 cases was thus 68%. In other words, 32% of patients with proved septicæmia (by positive blood culture) recovered, the highest rates of recovery being among those with a blood infection of anaerobic streptococci or *Bacillus coli*. Daily tests showed that streptococci did not multiply in the blood so rapidly as was formerly thought. There is usually no great increase until the last few hours of life.

## Peritonitis.

The total number of patients with peritonitis admitted during the whole period was 53, with eight recoveries. During the first period ending December 31, 1931, there were 31 cases—11% of the admissions.

The difficulty of diagnosis is marked, as the classical symptoms are absent in puerperal cases. The only symptoms in the 30 fatal cases out of the 31 cases just mentioned were: tympanites in 22, tenderness in 13, pain in 9, rigidity in 4, diarrhœa



in 4, vomiting in 5, and free fluid (clinical examination) in 2 cases. If any two of these signs or symptoms are present, the authors recommend a small abdominal incision, and if on immediate microscopic examination streptococci or pus cells are found in the smear from the exudate, they drain the peritoneal cavity. I have, however, seen a combination of the two most prominent symptoms mentioned in more than one patient after a tedious labour without any peritonitis.

In regard to the diagnosis and treatment of septic peritonitis, the special methods adopted by the late Arthur Nyulasy, when gynæcologist to the Perth Hospital, Western Australia, may be recalled, as they were original and remarkably successful. He recorded in *The Lancet* of October 9, 1915, a series of six consecutively successful cases treated by caecostomy, and his matured views were set forth in *The British Journal of Surgery*, July, 1917, under the heading of "Septic Peritonitis: Treatment by Caecostomy". His other papers on the subject were published in *THE MEDICAL JOURNAL OF AUSTRALIA*.

#### Urinary Tract Infections.

There were 146 urinary tract infections. Ninety-five were infections by *Bacillus coli* and 29 by other organisms (staphylococci, hæmolytic and non-hæmolytic streptococci, and *Bacillus proteus*). The urine became clear within two or three weeks in 11 cases when the patients were treated on a ketogenic diet.

#### Anaerobic Streptococci.

They (anaerobic streptococci) are frequently present, both in the mild febrile cases and (less frequently) in those in which there is no fever or any definite sign of infection of the tissues. Probably in many of these cases they are merely saprophytic invaders of the placental site or of devitalized tissues.

#### On the other hand:

These organisms have now been cultivated from the blood in no less than thirty-six cases of puerperal fever by the Queen Charlotte's Hospital staff in the last three years, and we believe it to be established beyond doubt that they are potentially pathogenic organisms, second only in importance to the *Streptococcus pyogenes*. The total number of cases infected by anaerobic streptococci was probably about 25% of all the patients admitted.

The failure to detect them in the past was due to the use of unsuitable blood culture media (Colebrook and others). In many cases two or more distinct types were present together in the uterine cultures. Their most constant pathological association was thrombophlebitis spreading upwards from the veins draining the uterus and vagina. "Repeated rigors are almost pathognomonic of infection by anaerobic streptococci."

#### Thrombophlebitis.

The authors stress the difficulty of diagnosis and the risks of operation in these cases, but add that:

By occasional successes in cases which had, prior to operation, seemed quite hopeless, we feel justified in continuing to treat by abdominal section and vein-ligature certain cases in which the diagnosis of spreading thrombophlebitis seems probable.

Bearing in mind their appalling mortality when treated expectantly, the late Arthur Nyulasy operated successfully in quite a number of these cases at the Perth Hospital and subsequently embodied his views on such cases in a paper entitled "Puerperal Infection: A Plea for Early Operation in Pelvic Septic Phlebitis" and published in *Surgery, Gynecology and Obstetrics* in March, 1920 (pages 265-268); and in another paper, entitled "Puerperal Infection: Ligature or Excision of Veins", published in *THE MEDICAL JOURNAL OF AUSTRALIA* on May 5, 1923, page 499, he gave convincing reasons why excision of veins is preferable to simple ligature in such cases, as, for example, where the affected vein "had become a tube of pus".

The *post mortem* findings of the Queen Charlotte's Hospital staff show that this had occurred "in many cases". Simply to ligate a vein so affected would be worse than useless. I may here point out that the conclusion of the Queen Charlotte's Hospital reporters that repeated rigors are almost pathognomonic of infection (and thrombophlebitis) by anaerobic streptococci should be helpful in arriving at a decision regarding operation.

The history of the nineteen anaerobic (streptococcal) septicæmic cases shows that these infections are particularly liable to follow difficult and exhausting labours involving internal manipulations. In not one of the first ten of these cases was there a normal spontaneous delivery.

#### Bacillus Coli Infections.

Abundant cultures of *Bacillus coli* from the urine were obtained in 113 cases. Abundant cultures from the blood and urine were obtained in six cases. Many of the 113 cases also gave an abundant culture from the uterus.

#### Mixed Infections.

In one case both hæmolytic and anaerobic streptococci were isolated from the blood; in another, anaerobic streptococci were found in a pelvic abscess, while hæmolytic streptococci were obtained from the liver and spleen of the same patient, in pure growth.

#### Staphylococcus Infections.

There were two cases of infection by *Staphylococcus aureus* and both were fatal.

#### Mortality Rate of Septicæmic Infections.

The mortality rate of septicæmic infections was as follows:

Hæmolytic streptococci in the blood: mortality, 86%.  
Anaerobic streptococci in the blood: mortality, 30%.  
*Bacillus coli* in the blood: mortality, 20%.

There appears to be some discrepancy between the mortalities of the various types of septicæmia dealt with in the original report, which will be evident on comparing the figures given on pages 1164 and 1174 of that report in *The Proceedings of the Royal Society of Medicine*. I find some difficulty in reconciling these two sets of figures. Two very striking facts already mentioned in the report may

be contrasted here. Haemolytic streptococci were found in 42% of all cases admitted, and the greatest number followed a normal delivery. Anaerobic streptococci were found in at least 25% of those admitted, and every one of the first ten cases of infection by these anaerobes followed a complicated labour.

The basis of treatment was the intermittent uterine and vaginal application of glycerine, with good nursing in the open air as much as possible, by which means a large proportion of the patients made a good recovery.

For want of space I have referred to but a few of the salient features of this most interesting and instructive report, but would like to express the hope that the authors, in a later contribution, may add to our debt of gratitude by giving a detailed account of the "289 cases admitted with a history of normal spontaneous labour, in many of which no vaginal examination at all" had been made. Such a report, I feel sure, would prove profoundly interesting and instructive.

#### THE POSITION OF THE URINARY BLADDER.

By H. FLECKER, M.B., Ch.M. (Sydney),  
F.R.C.S. (Edinburgh),  
Cairns, Queensland.

ACCORDING to Cunningham ("Text Book of Anatomy", Sixth Edition, 1931), "the urinary bladder is situated in the anterior part of the *pelvis minor*, above and behind the *symphysis pubis*". The various illustrations given show this organ in sections of all adults to lie at least partly behind the bony pubes. The view on page 1254 shows the full bladder of the male to lie mostly above the pubic crest and only a small part of it behind the pubic bones, but the view on page 1258 shows the distended female bladder for the most part behind the pubes.

According to the same authority, only in the new-born or young child is the bladder situated entirely above the pubic crest, and the illustrations of sections of the new-born in both sexes (page 1260) show this.

The foregoing corresponds to the description usually given in most, if not all, text books in anatomy. Nevertheless, with the patient lying on his or her back, a direct antero-posterior radiological view practically invariably shows not only the full bladder, but even the bladder which is almost empty in either sex to be wholly above the pubic crest. It is indeed rare to find any part of the bladder obscured by bone. Since the use of contrast material, particularly "Uroselectan", has become very general, the bladder is now regularly visualized on X ray films, and the discrepancy between the position of the bladder in these and that usually described by anatomists is very considerable.

What is the cause of this discrepancy? Are the X ray appearances misleading, although taken in the living subject, or is the method of fixing the viscera in the dead subject faulty, and does it not give, therefore, the true position of organs as they exist during life? Cunningham (page 1403) admits "that the form and position of the stomach in the living subject differ considerably from that which it presents in the cadaver".

If, then, anatomical sections give a false idea of the position of abdominal organs, this is of considerable importance, for instance, in deep X ray therapy. Holfelder has prepared a large series of charts for use in treatment of uterine carcinoma, based on ordinary anatomical studies, and it is quite likely that the use of such charts must lead to considerable error in locating the uterus.

These notes may probably stimulate some anatomists to correlate their work with the appearances noted in radiological studies. That this has been much neglected in the past is particularly evident in the tardy recognition by both radiologists and anatomists of the value of the X rays in the study of epiphyses *et cetera*.

#### Reports of Cases.

##### AN UNUSUAL DRUG ERUPTION.

By J. M. O'DONNELL, M.B., Ch.M.,  
Honorary Dermatologist, Fremantle Hospital;  
Honorary Assistant Dermatologist, Perth  
Hospital, Western Australia.

IN view of Sir E. Grahame-Little's recent article in *The British Medical Journal*, I think that the following case of possible bismuth deposits in the skin is particularly interesting.

Sir E. Grahame-Little drew attention to the occasional occurrence of eruptions like *lichen planus* following bismuth therapy which we occasionally see. However, in the literature at my disposal I have not been able to find any reference to actual deposits of the metal in the skin.

S.G., a male, aged twenty-seven years, came under observation in the early part of 1932 for a primary luetic infection. There is no history of secondary rash. After a course of "Neosalvarsan" (about four grammes) he was put on to intramuscular injections of bismuth hydroxide in oily suspension. He was receiving 0.15 gramme every week.

In a short time, about five weeks, he had to be given a "spell" because of gingivitis and bismuth deposits in the gums.

On resuming treatment he was given "Novarsenobenzol" injections again and after a suitable course was once more put on to bismuth, given intramuscularly. He soon developed gingivitis and perioral pigmentation. About this time, it appears, he went to the country, where he had a few intravenous injections of "Novarsenobenzol". After being in the country for some time he started gradually to develop black macules on his skin.

The appearance of these macules was gradual, and they did not all come out at once; the increase in numbers was gradual and progressive for several months. At no time during his treatment was there any symptom suggestive of sudden embolism from an accidental injection of bismuth into a vein in the gluteal region.

More recently, after a further intravenous injection of "Novarsenobenzol", I saw him, when he complained of pain in the finger tips and tenderness in the spots which showed a zone of erythema round each individual macule.

The distribution of the rash is as follows: Very marked in the ischio-rectal region, over the extensor surfaces of both elbows, about the neck and shoulder regions, round the hip regions, a few large macules scattered over the body, some perioral pigmentation. The mouth is clear at present (see Figures I and II).

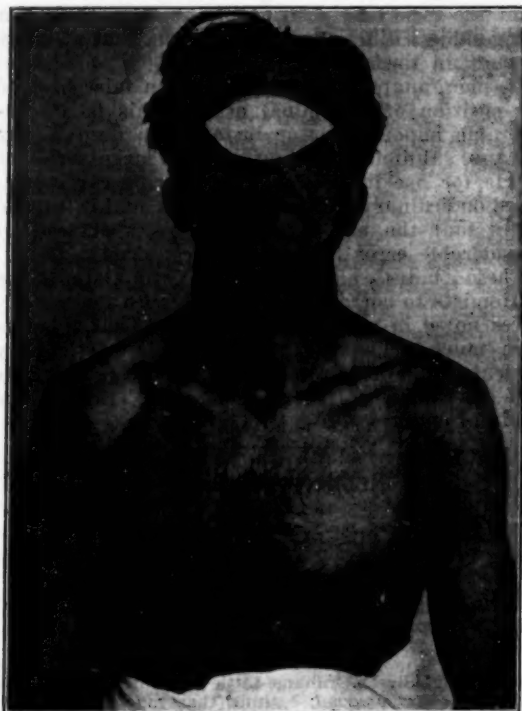


FIGURE I.

Showing bismuth deposits in the skin.

There is no palpable thickening or tenderness in the gluteal region, nor is there any history of either. The colour of the rash is that bluish-black so characteristic of bismuth.

A microscopic section of one of the macules revealed black possible foreign body deposits through the *cutis vera*. The arrangement was irregular, except for a tendency to be a little more numerous at the junction of the *cutis vera* and epidermis. There was little appreciable cellular infiltration. The deposits were irregular in shape and size, and none were globular; the appearance suggested fine metallic dust blown through the section.

Because of the evident "hardening" of the particles of bismuth, I am inclined to think that there will be a permanent "tattooing" of the skin.

The patient's general condition is not very satisfactory, as he is very neurotic and is suffering from chronic bronchitis, thought by Dr. Harke to be due possibly to bismuth deposits in the lungs. The patient does not react to the Wassermann test at present.

#### Acknowledgement.

I am indebted to Dr. Laurence Harke, of the Perth Hospital Clinic, for permission to publish his case; to Dr. Harry Lucraft for excellent photographs; and to Dr. Cohen and Dr. Clement, of the Pathology Department, for the preparation of microscopic sections.

#### Post Scriptum.

Owing to the unusual nature of the eruption, I sent a section of the skin to Dr. Roxburgh, of London. He expressed the view that the pigmentation was of arsenical origin and that the pigment deposits were melanin. He does not seriously consider bismuth as the cause and regards it as most unlikely.

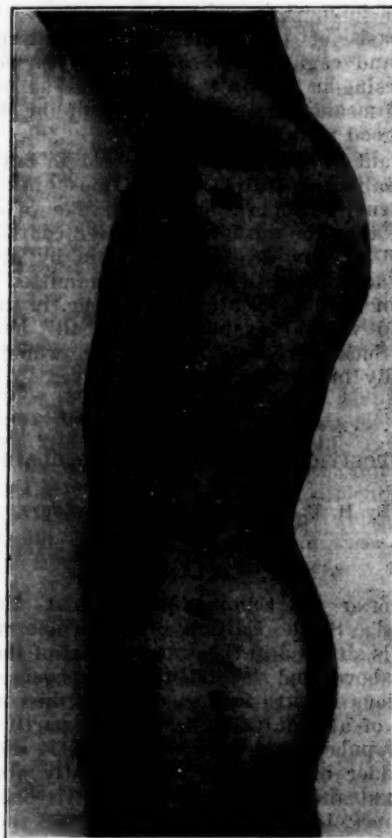


FIGURE II.

Showing bismuth deposits in the skin.

The inflammation in the pigmented spots following further injections of "Novarsenobenzol" is also very suggestive of arsenic as the cause of the eruption.

This inflammatory reaction has been observed by Dr. McGlashan and myself since I sent in the case report. Up to date the pigmentation shows no sign of spontaneously disappearing.

#### Reviews.

##### TREATMENT BY SUGGESTION.

DR. LEONARD INKSTER, as a disciple of Dr. Haydn Brown, in "The Treatment of Functional Nerve Cases by the Method of Neuro-Induction", has given a description of his method, together with his personal experiences.<sup>1</sup> The term neuro-induction is hardly descriptive of the technique,

<sup>1</sup> "The Treatment of Functional Nerve Cases by the Method of Neuro-Induction: An Essay", by L. Inkster, M.A., M.R.C.S.; 1933. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 80. Price: 3s. 6d. net.



which primarily aims at mental and physical relaxation as providing the correct atmosphere for the implantation of suggestive treatment. The patient is placed on a couch and told to lower his upper eyelid and hands at the same time. The procedure produces an automatic relaxation which leads to easy thinking. Positive suggestions leading to alleviation of symptoms are then given.

He regards two main processes at work in human beings. "The negatives make for difficulty, un-ease, disease; the positives for ease, power, health." Among the former are classed depression (self-pity), greed, self-consciousness, worry, obstinacy, helplessness *et cetera*. The corresponding positives would be self-congratulation, gratitude, altruistic thinking, ease, cooperation, power of self-help. In giving suggestion the direct opposite of the negative phase is stressed, and this tends to produce an uplift of the positives in general, a phenomenon termed positive circling.

The author is at pains to differentiate the state of relaxation from that due to hypnosis. Much of the curative value is ascribed to the "easy thinking" producing its own catharsis.

Although Dr. Inkster disowns Freudian leanings, it is apparent that the method in certain aspects is closely akin to treatment by free association. Illustrative cases are cited and good results are reported.

The essay is worthy of close attention by those practising psychiatry, but is of little interest to the general practitioner.

#### DERMATOLOGY.

THE fourth edition of "The Treatment of Diseases of the Skin", by Dr. Sibley, has just appeared thirteen years after the last edition.<sup>1</sup> It is a volume of 223 pages, with twenty-four photographic plates, printed on special paper, and is divided into three sections: (i) methods of treatment, (ii) skin diseases, (iii) prescriptions, perfumes, colouring materials, and index.

The section dealing with X ray therapy is vague and obsolete. The practical methods of production of X rays by various types of transformers and tubes are not mentioned. The system of dosage described is that of the pastille and fractions thereof, but we are given no hint as to what a pastille dose is or how it is measured.

The important question of filtration is not discussed in this present edition, although in the first edition of 1912 mention is made of the use of "a thin sheet or disc of aluminium". Radium therapy was not described in the first edition, but was in the second, issued in 1916. The section of 1933 is almost entirely the same as that of 1916.

Apparently the only types of applicator known to the author are: (i) flat applicators, the strengths of which are quoted in the obsolete radium bromide equivalents, which are no longer used; (ii) capillary glass tubes, now never used in ordinary practice; (iii) radium emanation collected in glass or metal containers. Modern metal tubes and needles have apparently not come within the purview of the author.

Ultra-violet rays receive attention, but only the air-cooled type of lamp is mentioned in the section, although later in the text the Kromayer lamp is advocated for the treatment of *alopecia areata* and *lupus vulgaris*. That this type of lamp is water-cooled the reader is left to discover himself.

Simplicity of classification of the various skin diseases has been achieved by the simple expedient of arranging them in alphabetical order.

The general lines of treatment of acne are well described, except that futile statements, such as "the sexual life should be regulated according to the laws of Nature and unnatural abstinences avoided", have no place in a modern text book. *Carcinoma cutis* merits only eleven lines. Early excision is suggested unless there is rapid improvement by full doses of X rays. Readers will be happy to

learn that large doses of arsenic have been advocated and that many cases have been satisfactorily treated by radium.

Voluminous details of the mercurial treatment of syphilis are provided. Poor new-fangled bismuth is fortunate in receiving a detailless mention. A comparison was made with the first edition; only minor additions have been made in the present one, but the reader is spared the unpleasant details of the inunction method. The arsenicals receive the same slight attention as they did in 1912. The outrageous statement is made that as "Neo-Salvarsan" is completely soluble and the solution neutral, it can be given in large doses without ill effect.

This book obviously cannot be recommended to readers.

#### INFRA-RED RAYS.

WITH the increasing proofs of the value of the ultra-violet rays in medical treatment there has been a natural tendency to investigate the therapeutic properties of other forms of light. Of these infra-red rays have been proved to be of value, more particularly in the relief of pain. In the second edition of "Therapeutic Uses of Infra-Red Rays" Dr. W. Annandale Troup gives a detailed account of these rays, the indications for their use in medicine, and the technique which he has found effective in his treatments.<sup>1</sup> In the preface to this book the author points out that the necessity of publishing a second edition to his work shows the interest taken in the infra-red rays. He offers as a further reason for republishing his book the changes in technique made necessary by experience.

Dr. Troup is to be congratulated on his book, which is short, concise, and readable. He is also to be congratulated on limiting the indications for treatment to a definite area. Unfortunately, many writers on physiotherapy have done harm by an unwisely attempt to find in the methods they advocate something approaching a cure-all. Those who have attempted to apply these remedies in accordance with such teaching have too often found themselves involved in failure and have had to cut out many diseases which these authors have professed to cure. Dr. Troup relies chiefly on the analgesic properties of the infra-red rays in such conditions as neuralgia, neuritis and *tic douloureux*, although he claims to have cured or relieved other complaints, such as the group of diseases commonly classed as rheumatic. In many diseases he has found the combination of infra-red and ultra-violet rays more efficacious than the use of either remedy alone.

To the increasing number of practitioners who are interested in physiotherapeutic remedies this book will prove helpful and interesting.

#### AN EXPERIMENT IN SEX EDUCATION.

"SEX EDUCATION IN SCHOOLS", by Theodore F. Tucker and Muriel Pout, is well worth reading by parents, teachers and physicians.<sup>2</sup> The authors gave a course of three lessons on sex hygiene to over 8,000 children aged thirteen *plus* in 111 schools in Wales. The subjects were "Keeping Fit Through Boyhood (or Girlhood)", "The Growth of New Life", and "Mind and Body". *Verbatim* reports of two of these lectures, as varied for boys and girls, and an outline of another lecture are given.

Such an experience qualifies the writers to deal with the supposed and real difficulties likely to be encountered in class instruction, and this they do very fully; yet they leave the impression that the difficulties are but slight and mostly imaginary. They truly say: "a subject which is openly discussed in class retains no longer the attraction of something whispered in secret".

<sup>1</sup> "Therapeutic Uses of Infra-Red Rays", by W. A. Troup, M.B., Ch.B., with foreword by Sir William Willcox, K.C.I.E., C.B., C.M.G., M.D., F.R.C.P.; Second Edition; 1933. London: The Actinic Press, Limited. Demy 8vo., pp. 90, with illustrations. Price: 6s. 6d. net.

<sup>2</sup> "Sex Education in Schools: An Experiment in Elementary Instruction", by T. F. Tucker and M. Pout, with a foreword by W. Brown, M.A., M.D., D.Sc., F.R.C.P.; 1933. London: Gerald Howe, Limited; Australia: Angus and Robertson, Limited. Crown 8vo., pp. 155. Price: 3s. 6d. net.

<sup>1</sup> "The Treatment of Diseases of the Skin", by W. K. Sibley, M.A., M.D., B.C., M.R.C.P., M.R.C.S.; Fourth Edition; 1933. London: Edward Arnold and Company. Demy 8vo., pp. 231, with illustrations. Price: 10s. 6d. net.

The experiment has certainly demonstrated that both parents and children desired that such teaching should be given and that no evident harm occurred from the instruction given. The questions asked by the children showed a serious interest in the subject and that "Please, how was I born?" was the focus of the questing mind.

The authors think that the teachers should be specialists in their subject and suggest that individual instruction by the family doctor has its own inherent dangers.

In spite of the writers' insistence on religious ideals, their lectures, as reported here, neglect the mysterious, the religious, the racial side of sex; they do not deal with its importance to the existence and the morale of nations and to the happiness of individuals and of families. They deal with detail rather than with principle.

### NEUROLOGICAL EXAMINATION.

We have before us the sixth edition of Dr. Monrad-Krohn's book on the "Clinical Examination of the Nervous System", being a compendium of selected and approved methods to be followed in the diagnosis of nervous diseases.<sup>1</sup>

At first glance the reader unacquainted with this book might think that if the prescribed ritual were to be minutely followed the task of examination would be so time-absorbing as to be impossible for the busy practitioner; and so it would be if the proceedings were to be taken from cover to cover. But this is not the way in which the book is intended to be used. We all know that even a moderately acquainted examiner can run the rule over a subject of ordinary nervous disease and disclose all salient features within a reasonably short time. What we may not realize is the difficulty of carrying in our heads details of unusual diagnostic technique which we have to apply when we meet extraordinary subjects. Then, as a memory refresher, the need and usefulness of such a book becomes apparent. Moreover, from its pages the student and young practitioner may not only gather a good working routine, but he may learn that there are right and wrong ways of performing such apparently simple tests as those to determine the state of the knee jerks or common sensibility or coordination of muscles.

In reviews of previous editions we have referred to the descriptive accuracy; we need only add that the present edition contains additions and amendments consistent with the progress of neurological learning.

### NEUROPATHOLOGY.

NEUROPATHOLOGY, in common with other medical sciences, continues to advance, and this has been accelerated by the application of the newer metallic stains and experimental methods of approach. Most specialists have perused maybe hundreds of monographs and have then picked up some book presenting in well balanced selections all their readings and much more. Such a book is Well's "Text-Book of Neuropathology".

The author appears to sense modern currents of thought and commences by issuing a warning against having too hard and fast rules as to what constitute normal and pathological histological pictures after common methods of fixation and staining have been used.

Cerebral chemistry and agonal disfiguration having been dealt with, our hopes are restored. (as regards trusting the evidence of anything we may see) by noting the author's description of eight varieties of pathological change in the ganglion cells in different states. Succinct but adequate chapters deal with modern conceptions of the rôle of the microglia and oligodendroglia in brain reactions, different types of demyelination, difficulties in

differentiating infective from vascular and degenerative phenomena, without neglecting many new conceptions on lesions in exogenous and endogenous intoxications, including alcohol, carbon monoxide and lead. As one would expect, much criticism is based on experimental work, the effect of prolonged ether anaesthesia on peripheral nerves for example.

Arteriosclerosis seems to be able to account for much that has commonly passed for more specific states.

The chapter on tumours is adequate, although often the author has to give both sides, as in the neurofibromata, where the "schwannoma" work of Masson and the French school are compared with the perineurial and endoneurial aetiology of the opposing school.

A useful appendix contains directions for fixing and staining tissues; it contains mostly those methods which, modified in the author's laboratories, have proved themselves. Judging by the numerous illustrations, apt and to the purpose, these methods have portrayed essential structures so well that microphotography has readily done the rest. Once read, one would wish to refer to this book constantly.

### RHEUMATOID ARTHRITIS.

The appearance of a second edition of Dr. Douthwaite's book on "The Treatment of Rheumatoid Arthritis and Sciatica" is clear evidence that it has been found to be of practical value.<sup>1</sup> Much of the matter has been either revised or rewritten, and methods of treatment introduced since the first edition was published in 1929 are fully discussed. Dr. Douthwaite is to be congratulated upon the clarity of his writing and on the temperate manner in which he sets his results before his readers.

The review of the aetiology is very well written and sets out very clearly the extent of our present day knowledge, dealing in turn with the rôles of focal infection, diathesis, and metabolic changes. These chapters should help to dissipate the rather prevalent idea that focal sepsis is the determining factor in all these joint affections.

The idea of dividing arthritis, for guidance in the method of treatment to be adopted, into three stages, though admittedly somewhat arbitrary, is a very useful one and should prove decidedly helpful.

The inclusion of sciatica in this edition is a new departure, but greatly enhances its value. The results that the author records of the treatment of this disabling condition by intrasacral injection and spinal manipulation are very convincing, and the careful details supplied for carrying out these procedures should prove very useful. We can quite confidently recommend this little book to every practitioner and can assure those who already possess the first edition that it is well worth their while to procure the second.

### SURGERY IN TABLET FORM.

"SYNOPSIS OF SURGERY", by E. W. Hey Groves, consists of 693 pages packed full of condensed surgery.<sup>2</sup> It is essentially a book of headlines, and therefore not for a leisure hour; but for refreshing the memory and recalling the points in any surgical condition it would be hard to beat. It is very complete in its detail for such a work and should prove an invaluable aid for revising work for examinations in the higher degrees of surgery. The additions in the tenth edition, embodying all the latest methods for diagnosis and treatment, make it thoroughly up to date. The arrangement of the subject matter makes it an easy book of reference.

The whole book tells us that it is the careful work of a master craftsman, who deals familiarly with proven methods, who has winnowed his grain carefully and presents it to us free of all chaff.

<sup>1</sup> "The Treatment of Rheumatoid Arthritis and Sciatica", by A. H. Douthwaite, M.D., F.R.C.P.; Second Edition; 1933. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 144, with illustrations. Price: 6s. net.

<sup>2</sup> "A Synopsis of Surgery", by E. W. Hey Groves; Tenth Edition; 1933. Bristol: John Wright and Sons, Limited. Crown 8vo., pp. 701, with illustrations. Price: 17s. 6d. net.

<sup>1</sup> "The Clinical Examination of the Nervous System", by G. H. Monrad-Krohn, M.D., F.R.C.P., with a foreword by T. G. Stewart, M.D., F.R.C.P.; Sixth Edition; 1933. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 253, with illustrations. Price: 7s. 6d. net.

<sup>2</sup> "A Text-Book of Neuropathology", by A. Well, M.D.; 1933. Philadelphia: Lea and Febiger. Medium 8vo., pp. 335, with illustrations. Price: \$5.00 net.

## The Medical Journal of Australia

SATURDAY, DECEMBER 16, 1933.

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### LEAD POISONING IN INDUSTRY.

THE papers by Dr. George Willcocks and Dr. Charles Badham published in this issue are of more than usual importance. Lead poisoning is so insidious in its onset and so disabling in its effects that medical practitioners will do well to study these papers with particular care. Medical practitioners in Queensland are accustomed to look for signs and symptoms of lead poisoning; in other places this habit has not perhaps been formed. Practitioners may learn from these papers the importance of blood changes as a deciding factor in diagnosis; and, if they do this, the papers will not have been published in vain. Important, however, as the recognition of lead poisoning in private practice is, the industrial aspect is a matter of much greater concern, for it is in industry that prevention can be practised, and it is from workers in lead that many of the cases of lead poisoning seen in private practice are drawn. Attention should therefore be directed to two statements of these authors. Dr. Willcocks states that it is a very haphazard state of affairs when twenty or thirty men have to be poisoned before adequate precautions are taken. Dr. Badham writes: "The obvious duty of an industrial hygienist is the detection of lead poisoning and its prevention. It

should be practicable to conduct lead processes without causing lead poisoning, but at present this ultimate boon is not in sight."

There is no gainsaying the fact that the prevention of lead poisoning in many industries has been neglected, in spite of the work done more than a decade ago by the Technical Commission of Inquiry that made its investigations at Broken Hill. Were it not so, Dr. Willcocks would not be able to publish in his paper results of investigations made by Dr. Badham and the officers of his department, showing such a large number of affected persons. Nor would Dr. Duhig have found lead poisoning so prevalent at Mount Isa. (Dr. Duhig's findings are described in a special abstract in another place in this issue.) There is nothing new about the recommendations made at Mount Isa in regard to ventilation, cleanliness and so forth; it is extraordinary that at this stage in industrial history such recommendations should have to be made. Still, lead poisoning exists, and this brings us to Dr. Badham's remarks about prevention.

Dr. Badham states that the duty of an industrial hygienist is detection of lead poisoning and its prevention. We would reverse the order of the words. If we seek prevention first, most other things will be added to us. To seek prevention of disease in industry implies a wide-awake state; it implies an understanding of the chemical and physical processes of the industry; and it also demands inquiry into the pathological processes induced in the human body as a result of industrial hazards. If we seek prevention with singleness of eye, unavoidable disease, if there be unavoidable disease, will be discovered in its early stages and steps may be taken to treat the sufferer and, if necessary, to find employment for him in another industry. Dr. Badham would, of course, be the first to agree with these statements, for the whole activities of his Division of Industrial Hygiene are directed towards this end. We must therefore agree with Dr. Duhig when he states that we have at Mount Isa an opportunity of making a contribution to industrial hygiene of the highest possible value. Dr. Duhig rightly estimates the value of any service, especially that of public health, not only



on the efficiency with which it deals with immediate problems and the value of immediate results thus obtained, but also and most particularly on the contribution that it makes to human knowledge and human welfare. It would be of immense value to Queensland workers if the 93% of illness mentioned by Dr. Duhig could be eliminated; it would be of even greater value if research into the whole question of lead poisoning were undertaken at such a promising centre as Mount Isa. There is one of the suggestions of Mr. Watson and Dr. Duhig to which exception might be taken, and that is the suggestion that the chairman of the proposed board, who is to undertake the research, should be appointed for a period of only two years, renewable for a further period. Research on such a difficult problem will not easily yield results of great moment. If a research worker even of the widest experience were appointed, it would not be fair to expect him to justify his appointment by producing results in a period of two years, for fear of losing his position, when perhaps a much longer period would be required. If reappointment were in the hands of people who were not lacking in understanding, this difficulty would not arise. However, if the Government of Queensland agrees to establish research at Mount Isa, it will be doing something to promote the health of lead workers in every place.

### Current Comment.

#### HYPERTENSIVE ENCEPHALOPATHY IN NEPHRITIS.

OF latter years the syndrome of uræmia has been subdivided into two types of distinct ætiology. One is called latent or toxic uræmia and represents the symptoms and signs that develop solely as a result of the cessation of renal activity, for example, when a single kidney is removed or both ureters are tied at operation. The other is called sthenic uræmia, and consists of the secondary effects of prolonged renal failure on the arterial system, and especially upon intracranial hydrodynamics; it causes headache, coma, convulsions, amaurosis and other evidences of diffuse or focal disturbances in the brain, together with hypertension. A new name, "hypertensive encephalopathy", has been coined by Oppenheimer and Fishberg to describe these sequelæ. A chronic nephritic during a crisis shows,

of course, clinical signs of both types, while sufferers from essential hypertension, encephalopathy and eclampsia may at times show exactly similar signs to those seen in the sthenic type. Horace Evans states, however, that frank signs of hypertensive encephalopathy are uncommon in nephritis.<sup>1</sup> He reports nine instances, seven in acute nephritis and two in chronic nephritis, collected over a period of four years from ninety patients with acute and seventy patients with chronic nephritis. Arterial hypertension was present in every case, an additional rise frequently preceding the actual attack of encephalopathy. Headache, puffiness of the face, anæmia, and coma of varying grade were present in every case. Vomiting, of the cerebral type and frequently severe, was noted in seven patients, fits in six, amaurosis in five, retinal arterial degeneration in four. In one instance Evans observed the development of a gross neuroretinopathy, as he terms it, in twenty hours. One patient was blind for ten days, but completely recovered his sight. Prominence of the veins about the head and neck, neck rigidity, pyrexia, slowing of respiration rate and cataleptic phenomena were also recorded. Cerebro-spinal fluid pressure was raised in seven of the nine patients. The interesting feature was the result of blood urea estimations and renal function tests performed during the height of the attacks. They showed little or no further renal deterioration, and usually the amount of urine was increased. This is to be correlated with the appearance of these phenomena in patients in whom no renal degeneration is known to exist, for example, those suffering from essential hypertension, eclampsia, and the preliminary rise in blood pressure with what is well known to occur in *eclampsia gravidarum*.

The brain shows no evidence of focal lesion when examined at necropsy. "Cerebral œdema" is an unsatisfactory explanation, as no such symptoms occur in nephrosis nor in many other instances in which œdema of the brain is to be found. Volhard believes this œdema to be a secondary result of cerebral ischaemia from arteriolar spasm, an idea which is consistent with the rapid onset and disappearance of the symptoms, and direct observations of spasm of retinal arteries during an attack of eclampsia. Evans himself failed to observe any spasm while watching the fundi of his own patients.

The diagnosis of this state is relatively simple. Lumbar puncture will assist in the differentiation from subdural hæmorrhage or meningitis. True epilepsy and cerebral tumour must also be eliminated. The diagnosis between hypertensive encephalopathy and true uræmia is important from the point of view of prognosis, since none of Evans's patients with acute nephritis and cephalic symptoms died. The amaurosis usually clears up within hours or days. Frequent and severe fits are of ill omen, as heart failure is apt to follow with little warning. Therapy must be directed to

<sup>1</sup> *The Lancet*, September 9, 1933.

improving the cerebral circulation; venesection and lumbar puncture are two methods of undoubted value, and bleeding should be proceeded with unless there is a very severe anaemia. Evans recommends that the volume of blood removed should be 400 cubic centimetres for children of ten to fourteen years, and 600 cubic centimetres for adults. The reason for the beneficial action of venesection is not clear, as the immediate fall in blood pressure is often very slight. While lumbar puncture is usually successful, the results are occasionally disappointing, even in the presence of raised pressure. Intense headache is, however, considerably relieved. Evans recommends a slow drainage, preferably following the venesection. The intravenous use of hypertonic saline or magnesium sulphate solution has a place here, but unfortunately these substances increase the blood volume, a potential danger in acute nephritis. Morphine should be used in every instance, and cases of hypertensive encephalopathy which fail to respond to the above methods of therapy are rare.

Evans's case reports are very complete and substantiate the findings and conclusions of American writers on this subject. Most of his views are generally accepted, but misconceptions are plentiful as to the frequency and prognosis of hypertensive encephalopathy. It is surprising to find that the syndrome was prominent in only nine patients of one hundred and sixty suffering from acute or chronic nephritis. The results of the treatment of Evans's nine patients are reassuring to the physician charged with the care of a nephritis patient who suddenly manifests the symptoms of hypertensive encephalopathy.

#### ARTIFICIAL PNEUMOTHORAX.

THE induction of artificial pneumothorax is perhaps the most valuable single measure used in the treatment of pulmonary tuberculosis; yet a great deal remains to be learnt of its mode of action. Presumably its most important effect is the resting of the diseased lung. When the lung is rested, apparently lymph stasis is produced, hence prevention of the spread of the disease. The function of the lymphatic system is of very great importance in tuberculosis; and it has been suggested that the actual type of lymphatic structure may be a factor in determining the type of disease produced by tuberculous infection. There is enough known and conjectured to demand at least that careful investigations should be continued. Reginald Hilton has recently conducted an interesting series of experiments with a view to noting the effects of artificial pneumothorax on the pulmonary lymphatic vessels of rabbits.<sup>1</sup> He injected a suspension of fine carbon particles into the trachea, and immediately induced pneumothorax; some sterile liquid paraffin, in addition to air, was injected into the pleural cavity to lessen the frequency of refills. The twelve animals used in the experiments were killed after six months

or less. The collapsed lung was found to contain much more carbon than the opposite, or working, lung. Two control animals, killed four months after the injection of carbon, appeared at a superficial examination to have practically none left in their lungs; but microscopic examination revealed that many alveoli still contained particles. The most interesting feature was the histological appearance of the collapsed lung. The lymphatic vessels were grossly dilated and contained masses of carbon; the quantity was greatest in the vessels near the hilum. The alveoli also contained more carbon than those of the working lung. The great dilatation of lymphatic vessels was seen only in the collapsed lung. There can be little doubt that in these experiments the collapse of the lung prevented the rapid removal of carbon particles; presumably the condition of the lymphatic vessels had something to do with this delay.

Hilton remarks that it is difficult to conceive of any cause, other than obstruction, for the dilatation of lymphatic vessels in a healthy lung. If there were no obstruction, collapse of the lung would be expected to be accompanied by collapse of its contained lymphatics as well as its capillary blood vessels. He suggests that the obstruction is caused by some kinking of the main lymphatic vessels near the root of the lung. Whatever its cause, it evidently takes effect immediately pneumothorax is induced, as the difference in carbon content of the two lungs is noticeable within a few weeks. The difference is more than could be accounted for by the difference in the rate of elimination along the air passages. Lymphatic dilatation is also observable in human lungs that have been in a state of collapse for some time. We may reasonably conclude that pneumothorax causes lymphatic stasis.

It is known that there are no lymphatic vessels in the walls of the air spaces distal to the *ductuli alveolares*. Hilton remarks: "Such tubercle bacilli as gain admission to the beginnings of the lymphatic plexus do so within a phagocyte, as carbon does." It seems probable "that the transference of tubercle bacilli along the lymphatics will be slowed by lymphatic stasis and the chance of metastatic infection will be diminished". He also points out that the induction of artificial pneumothorax is often followed by a rapid and striking improvement in the patient's general condition. This improvement occurs too soon after the commencement of treatment to be due to any alteration in the actual tuberculous process; he suggests that it may be due to lymphatic obstruction and consequent prevention of the escape of toxins into the circulation.

Dilatation of the lymphatic vessels in a collapsed lung is a remarkable phenomenon. Hilton's attempted explanation of its cause is interesting but inconclusive; it would be more readily accepted if an obstruction could be demonstrated. Hilton's work is important; it is to be hoped that he continues it, with the object of learning more of the histological effects of pneumothorax and the part played by the lymphatic system in tuberculosis.

<sup>1</sup> *Proceedings of the Royal Society of Medicine*, July, 1933.

## Abstracts from Current Medical Literature.

### PHYSIOLOGY.

#### The Activity of the Muscle of the Human Uterus.

HUMAN uterine muscle removed at operation supplied the material for the following observations by J. M. Robson (*Journal of Physiology*, July, 1933). Strips of muscle were suspended in oxygenated Ringer-Locke solution at 37° C. and records of their contractions were obtained. Muscle from a uterus eight weeks pregnant reacted to fairly large doses of vasopressin—0.5 unit per 100 cubic centimetres of solution—but was unaffected by oxytocin in a concentration of one unit per 100 cubic centimetres. A strip removed at the twelfth week contracted on the addition of two units of vasopressin to the standard 100 cubic centimetre bath, while a similar dose of oxytocin was without effect. As a rule, muscle taken during the early stages of pregnancy showed a comparatively low reactivity to oxytocin. Between the seventeenth and thirtieth weeks there was an increase in the response to oxytocin, no samples showing a complete lack of reactivity to large doses. Muscle obtained during the period between the thirty-third and fortieth weeks of gestation responded to doses of oxytocin ranging from 0.1 to 0.01 unit. In five cases muscle was removed during labour. Three of these samples responded to 0.01 unit, while another contracted on the addition of 0.003 unit of oxytocin. The reactivity of muscle from the body of the uterus appeared to be greater than that of strips from the lower uterine segment. All samples of muscle removed during labour reacted to histamine in low concentration, for example, one in 10,000,000 to one in 100,000,000. There appears to be no correlation between the reactivity to histamine and to oxytocin. Ergotoxin in a concentration of one in 10,000,000 caused contraction in all strips removed during parturition. The action of ergotoxin is sustained and usually persists after the solution bathing the muscle is changed.

#### Carotid Sinus Reflexes.

J. J. BOUCKAERT AND C. HEYMANS (*Journal of Physiology*, July, 1933) have reinvestigated the question of the relative parts played by the reflexes arising in the carotid sinus and the cerebral blood flow respectively in the control of general blood pressure, cardiac rate and respiratory activity. In dogs low cerebral blood pressure and diminution in the flow of blood through the brain produced by occlusion of the efferent branches of the common carotids, occlusion of the vertebral arteries or of the denervated common carotids are without effect on the vasomotor, cardio-regulatory

and respiratory centres. On the other hand, if the pressure in the carotid sinus be low, the vasomotor and respiratory centres are stimulated, even when the cerebral blood pressure and blood flow are increased as a result of the vasomotor reflexes. Conversely, high carotid sinus pressure associated with decreased cerebral blood pressure and flow depresses the activity of the vasomotor and respiratory centres. The cerebral blood supply is passively dependent on the general and cephalic arterial pressure. In the case of decreased cephalic pressure the blood is deviated by means of the carotid sinus reflexes from the peripheral to the cephalic circulation. Conversely cephalic arterial hypertension causes reflexly a deviation of the blood from the cephalic to the peripheral circulation. In the reflex regulation of blood flow the arteries of the central nervous system take no active part, but act merely as a passive conducting system. The tonus of the vasoconstrictor centre, which is normally partially inhibited by the effect of the normal blood pressure on the receptor field of the sinus and aorta, is maintained chiefly by the tension of carbon dioxide in the arterial blood. The respiratory centre is more directly sensitive than the vasomotor centre to chemical changes in the blood. This sensitivity is not to moderate degrees of anoxæmia, but to the carbon dioxide content of the blood. After section of the sinus and aortic nerves the general blood pressure increases and remains chronically high, but the activity of the respiratory centre, which is at first increased, returns progressively to normal and may later be less than normal.

#### Cutaneous Pain.

SIR THOMAS LEWIS AND W. HESS (*Clinical Science Incorporating Heart*, Volume I, page 39) have shown that, apart from "dull" pain produced by pressure in its deeper layers, the skin is capable of giving rise to pain of one quality only, and that different types that have been described vary chiefly in their duration. Skin injured sufficiently in any way assumes a susceptible state in which the pain nerve endings are hypersensitive. The skin is then hyperalgesic and its pain threshold to heat stimuli is lowered. When in this state, skin usually displays spontaneous pain when its temperature is raised to about 34° C. Venous congestion, however produced, often causes pain if the skin is in this state, apparently because of stretching of the skin by vascular engorgement. The susceptible state is not the immediate result of an injury, but appears to be associated with the ensuing inflammation. It is caused by the liberation of some substance from the injured tissues which acts on the pain nerve endings. When skin in the susceptible state is rubbed, pain develops after a latent period. Simple occlusion of the circulation may similarly cause pain by allowing the local concentration of the stimulating

substance to rise to threshold level. The mechanism of the production of pain during actual stimulation of the skin is distinct from that responsible for the pain as an after-effect of injury. Itching and pain are distinct phenomena, the former being associated with the triple response, the latter with a later stage of inflammatory reaction. It is suggested that the vasodilatation accompanying the susceptible state is due not to a histamine-like material, but to some other vasodilator substance.

#### Carotid Sinus Polyuria.

S. JANSSEN AND J. SCHMIDT (*Archiv für experimentelle Pathologie und Pharmakologie*, September, 1933) have found that in rabbits and dogs, when both carotid sinuses are occluded from the circulation, there occurs after a latent period of one to two hours a polyuria which may persist for several hours. Simultaneously with the polyuria there is an increase in the absolute excretion of chloride, accompanied by an increase in the concentration of urinary chloride. In certain experiments the concentration of sodium chloride in the urine rose to 1.5%. Some animals exhibited only an increased chloride excretion without polyuria. If the carotid sinuses are occluded for twenty-four hours and then the circulation through them is once more established, there follows a diminution in the volume of urine and a fall in the absolute and relative chloride excretion. These effects are reflex in origin and are not dependent upon alterations in the cerebral blood flow consequent upon occlusion of the carotid arteries, for previous section of the carotid sinus nerves prevented the polyuria.

### BIOLOGICAL CHEMISTRY.

#### The Bactericidal Substance in the Urine of Patients Receiving a Ketogenic Diet.

ALBERT THOMAS FULLER (*Biochemical Journal*, Volume XXVII, Number 4, 1933) has isolated and identified the bactericidal agent present in the urine of patients receiving a ketogenic diet. For nine months the ketogenic diet was given to patients with pyelitis and cystitis at the isolation block of Queen Charlotte's Hospital. Routine examinations of the pH aceto-acetic acid content and *Bacillus coli* inhibiting power of the urine were carried out at frequent intervals on the patients under treatment. Methods for carrying out these examinations are given in detail. The principal factor inhibiting the growth of bacteria in the urine was found to be L-β-hydroxybutyric acid (levo-β-hydroxybutyric). The estimation of this acid, compared with the estimation of aceto-acetic acid, is a very lengthy process, and since the concentrations of both these acids increase with the intensity of the ketosis, the aceto-acetic



acid content of the urine is a good guide to the progress of the patient. The inhibitory power of the  $\beta$ -hydroxybutyric acid was found to increase considerably as the reaction of the urine became more acid, and the practical point that emerges from this piece of research is that acidifying salts should be given at the same time as the ketogenic diet, since the greater acidity helps moderate concentrations to become effective. The bacteriostatic power of other organic acids was also determined, and it was found that they too inhibited the growth of *Bacillus coli* in normal urine at pH 5.5. Aceto-acetic acid and acetone were found to be much weaker in their action than the hydroxybutyric acid, and since they are present in ketonic urine to a much smaller extent than the latter acid, they contribute only a small portion of the total activity.

#### The Determination of Calcium in Blood Serum and Cerebro-Spinal Fluid.

ARTHUR HAROLD TINGET (*Biochemical Journal*, Volume XXVII, Number 2, 1933) concludes that owing to errors associated with adsorbed organic matter and incomplete precipitation, calcium estimations by means of potassium permanganate titration or acidimetric methods effected on non-deproteinized serum or cerebro-spinal fluid cannot be relied upon to give absolute values or values such that significance can be attached to minor variations. The errors associated with these methods can be successfully eliminated by adopting the method of Halverson and Bergel, as modified by Peters and Van Slyke, including the Halverson and Bergel washing with removal of the mother liquors by suction, as modified by Van Slyke and Sendroy. Fatty and opaque sera can be successfully analysed by this method.

#### Quebrachitol as a Sweetening Agent for Diabetics.

ROBERT ALEXANDER McCANCE and ROBERT DANIEL LAWRENCE (*Biochemical Journal*, Volume XXVII, Number 4, 1933) have issued a report on quebrachitol. This substance, which chemically resembles inositol, was found to have similar physiological effects. When taken by mouth, it failed to relieve hypoglycæmia, did not raise the blood sugar or lead to the deposition of glycogen in the liver. Quebrachitol was not nearly so sweet to the taste as cane sugar, and two or three times the amount was found to be necessary to obtain the same effect on the palate and in such amounts on several occasions it produced either colic or diarrhoea. These workers do not recommend its use as a sweetening agent.

#### The Role of Tryptophan in Blood Development.

ROBERT SAXELBY ALCOCK (*Biochemical Journal*, Volume XXVII,

Number 3, 1933) has carried out experiments to determine whether the lack of tryptophan, and of tryptophan alone, does really produce anaemia, as Fontès and Thivolle stated, and whether rats, already anæmic, can recover from their anaemia while on a diet lacking in tryptophan. This investigator found that rats kept on a tryptophan-deficient diet over prolonged periods failed to develop anaemia and that young rats suffering from milk anaemia recovered from this anaemia independently of the presence of tryptophan in the diet. This recovery from anaemia of tryptophan-starved animals is difficult to fit in with the idea of the utilization of this amino-acid in hæmatin building. The results of other workers are explained by the fact that the anaemia may well have been due to the lack of some other dietary constituent besides tryptophan, and the results of work involving injections are explained on the basis of a stimulating action of tryptophan on the blood-forming organs. Alimentary tryptophan could not be expected to exert such an effect, as excess of this amino-acid in the portal system is converted into kynurenic acid in the liver. The author quotes the work of Matsuoka and Nakao, who showed that 2-methyl-tryptophan, while not able to replace tryptophan in the diet, would, when injected, nevertheless accelerate the recovery from phenylhydrazine-induced anaemia.

#### A Spectrographic Analysis of the Metallic Content of Meconium.

JOSEPH HAROLD SHELDON and HUGH RAMAGE (*Biochemical Journal*, Volume XXVII, Number 3, 1933) have investigated by the spectrographic method twenty-four specimens of meconium from twenty-four healthy full term infants. Lines due to sodium, potassium, calcium, magnesium, copper, manganese and iron were recognizable in all. No opinion in regard to the presence of zinc is given, as the presence of this metal is difficult to determine by this method. The most remarkable features of the spectrograms were the strength of the lines due to manganese and to a less extent those due to copper, and the weakness of the lines due to iron. The reason for the constant over-supply of manganese to the fœtus is obscure, as no fœtal store of this metal is built up in the liver, as is the case with iron and copper; but these investigators regard it as possible that while manganese is not stored in the fœtus, a constant supply is necessary for fœtal growth, and it is again steadily excreted. The weakness in the iron lines was as striking a feature of the spectrograms as the strength of the manganese, and the results obtained suggest that at least under the social conditions of town working class life the mother is able to supply the fœtus with an excess of all metals except iron. Either she is not able to supply iron or the avidity of the fœtal tissues for this metal is so

great that almost all the iron received from the mother is retained and any excreted into the gut is reabsorbed. The quantities of the metals found indicated considerable variations in the mother's power to supply them; in one case there was a high percentage and in another a low percentage of all metals. Such cases were, however, rare.

#### The Effect on the Blood Sugar and Blood Phosphate of Ingested Creatine.

REGINALD STEPHEN STACY (*Biochemical Journal*, Volume XXVII, Number 3, 1933) in a series of experiments on normal and diabetic patients estimated the blood sugar and plasma inorganic phosphate before and after the ingestion of creatine. The blood sugar was found to vary considerably during the course of an experiment, the variations being larger in diabetics than in normal persons. There was no evidence, however, of a tendency for it to rise or to fall after creatine in either type of persons, and the averages for both show practically no change. There was a tendency for the phosphate and creatine to vary together, a rise in the plasma creatine being accompanied by a fall in the phosphate, but the fall in the phosphate was not proportional to the rise in the creatine. No definite differences were to be seen on comparing the normal persons with the diabetics. Although the changes obtained were small, the author considers that the agreement of the results eliminates the possibility that they were chance effects, and draws the conclusion that a small drop in plasma inorganic phosphate follows the ingestion of creatine, but that this is not always constant in amount and is probably influenced by some unknown factor.

#### The Vitamin B Complex and High Protein Diets.

F. T. G. PRUNTY and M. H. ROSCOE (*Biochemical Journal*, Volume XXVII, Number 3, 1933), working with rats, have not been able to find any relation between the protein intake and the vitamin  $B_1$  requirements. Excess protein in the diet was found to have an indirect depressing effect on growth, since it caused a reduced food intake and this resulted in a lowered vitamin intake when the vitamins were incorporated as a percentage of the diet. The intake of vitamin  $B_1$  directly affected the rate of weight increase independently of any effect on the appetite, and growth was constant for any given dose of vitamin  $B_1$ , whereas the calorie intake varied with the food consumption within wide limits, according as to whether the diet had a high or low protein content. A high protein diet was not found to affect the incidence of the dermatitis characteristic of vitamin  $B_2$  deficiency. These investigators also found that hypertrophy of the kidneys of the rats on a high protein diet was independent of the vitamin  $B_2$  supplied.

## Special Abstract.

### LEAD POISONING AT MOUNT ISA, QUEENSLAND.

A REPORT into the incidence of lead poisoning at Mount Isa, Queensland, has recently been presented to the Parliament of Queensland. The report is signed by Mr. John A. Watson, F.I.C.A., Insurance Commissioner, and Dr. J. V. Duhig, Government Pathologist. The résumé of the chief features of this report should be read in conjunction with the leading article in this issue and the papers by Dr. G. C. Willcocks and Dr. C. Badham.

The terms of reference of the investigation were as follows:

1. Prevention or diminution of known lead hazard which assumed special significance during the year ended 30th June, 1932, and has not lessened in the present year.

2. (a) Medical certificates accompanying Workers' Compensation claims for incapacity by lead poisoning. (b) Medical investigation necessary to provide that in all cases medical certificates could be relied on for prompt payment of claims.

3. Settlement of compensation claims to the satisfaction of the worker, the employer, and the Insurance Commissioner.

From an inspection of the plant and from graphs of atmospheric pollution by lead it was evident that lead hazards of a high order definitely existed at certain points in the process of smelting. A test was made of claims for compensation during the half year ended December 31, 1932, and it was found that the workers affected were in the majority of instances employed in and around the smelter plant. Thirty-three cases of lead intoxication were found, and this number represented 22.49% of the total employees who were suffering from lead intoxication.

In the medical section of the report Dr. Duhig points out that prior to June 30, 1931, there were only eight claims for compensation on account of lead poisoning in Mount Isa. It was about this time that the plant went into production. Since then nearly 220 claims have been made. (Dr. Duhig's examination was made in May, 1933.) Since July 1, 1932, to March 1, 1933, the actual places of employment of workers claiming compensation have been recorded. These records show that the mine is responsible for 18.5% of the total claims for that period, the mill for 14.8%, and the smelting works for 66.6%; in other words, the smelters cause twice as much lead poisoning as the mine and the mill put together. The crude figures for the period are 25, 30 and 90 respectively out of a total of 135 claims for that period.

From July, 1931, to May, 1933, inclusive, 189 workers were admitted to hospital; some of these admissions were due to a recurrence of previous symptoms. These figures do not represent the total invalidity due to lead poisoning, since many employees suffer from the disease in a minor degree, are medically treated and cured, and return to work.

Dr. Duhig refers to one factor which, in his opinion, cannot be ignored. It was the stated policy of the management to employ only young healthy men without any previous experience of lead mining or smelting. Very few of the shift bosses are experienced miners. Dr. Duhig states that to inexperience and the resulting lack of care much of the incidence of plumbism at Mount Isa must be attributed.

Dr. Duhig goes on to discuss the results of examination of 1,892 employees. These examinations were made periodically. They represent the ordinary routine medical examination of employees. The examination was partly clinical and partly pathological. Smears of blood were examined and the blood examination was made quite independently of the clinical examination. The technique of the blood examination is described by Dr. Duhig as extremely good. He also adds that the data had the further statistical value that they were compiled from the private records of the medical practitioner who collected them without any idea that they would ever have to be made available to an independent outside authority. Of

the total number of 1,892 employees examined, 518, or approximately 27%, showed some slight or mild degree of lead poisoning. The serious cases numbered forty. Most of the slighter degrees of poisoning would have been missed or would have come to light at a much later date had periodical examinations not been made. The figures are regarded by Dr. Duhig as showing two things. The first is that a definite hazard exists, and the second is that serious illness can be prevented in 93% of the workers who are or are about to be affected.

In discussing the question of diagnosis, Dr. Duhig states his belief that lead poisoning does not exist without changes in the blood. He also holds that excretion of lead in the urine is evidence of lead absorption and of lead in the circulation. He devotes considerable space to the consideration of the views of various authorities on punctate basophilia and states his conviction that proper examination of the blood and especially a study of the morphological changes of the red cells by a person of great experience and patience are absolutely essential to the diagnosis of plumbism. He expresses the view that compensatable disability in acute cases does not exist unless punctate basophilia has been demonstrated by a suitable standard of technique to the extent of at least one thousand coarsely stippled cells per million. Less degrees of change are to him of importance only in so far as the early diagnosis of the condition is concerned.

Reference is made by Dr. Duhig to the work of Taylor and Tannahill, and he states that excretion of lead in the urine above a certain level, probably about 0.05 milligramme per litre, is evidence of exposure to lead hazard. "The fate of absorbed lead is to be deposited in the bones, where it does practically no harm. It is only the circulating lead that damages the tissues. No definite amount can be laid down as indicative of severe damage, since a large number of estimations goes to show that there is no constant relationship between lead intoxication and lead output, but taken in conjunction with exposure, both as to intensity of hazard and duration, and with other data such as clinical signs and blood alterations, estimations of this kind should sometimes prove of great value."

#### Recommendations.

The recommendations accompanying this report are placed in two groups. The first includes those made by Mr. Watson and Dr. Duhig, and the second group includes the purely medical recommendations made by Dr. Duhig alone. These two groups of recommendations must obviously be read together.

The recommendations of the first group include the posting of a warning notice in any place where any lead process is carried on so as to cause dust or fume or gas in the atmosphere. The text of the suggested warning notice is reproduced in Dr. Willcocks's paper in this issue. Recommendations are made regarding the prevention of the emission of flue dust from smelter stacks; the ventilation of tapping floors; the protection of persons entering furnaces *et cetera*; the ventilation of buildings, works and other places where lead processes are carried on; the maintenance of roads, pathways and yards so that dust shall not arise; the provision of overalls and close head covering; the use of respirators; the provision of meal places so that they shall not be exposed to dust or fumes from any manufacturing process; the provision of drinking water that is not contaminated by dust. It is proposed that restrictions shall be placed on the number of hours worked by employees in certain places; that every person for whom a respirator is provided shall wear it; that no food or drink, except a non-alcoholic drink, shall be consumed where a lead process is carried on; that no person shall make cigarettes or cut or roll tobacco in any place where a lead process is carried on.

The most far-reaching recommendation is that *The Workers' Compensation Acts, 1916 to 1929*, shall be amended to deal specifically with claims for incapacity by lead poisoning at Mount Isa and with medical certification of such incapacity. It is suggested that the Government should appoint a medical board consisting of three duly qualified medical practitioners, one of whom is to be nominated by the management, one by the employees'

unions, and the third to act as chairman. The chairman, it is suggested, should be located at Mount Isa and be nominated by the Government. The functions of the proposed board are set out fairly fully. The appointments are to be for two years and to be renewable for like periods. The members of the board are to exercise the powers and perform the duties of certifying medical practitioner or medical referee in respect of workers incapacitated by lead poisoning or lead intoxication while employed at Mount Isa mines. The board is to have power to determine the period of incapacity of any worker. It is suggested that the chairman shall receive a salary of £1,000 *per annum* net, and that the other two members of the board shall each receive an honorarium of £50 *per annum*.

In his separate report Dr. Duhig suggests that the chairman of the proposed board should be a highly qualified person and that he should be able to undertake research. "He should be a man of long experience and expert in all ordinary laboratory proceedings, particularly in so far as blood investigations are concerned, most especially those concerned with the detection of plumbism. He should also be capable of making lead estimations in body fluids and tissues, and in general he should be capable of planning and pursuing a very extensive course of research into the incidence and effects of lead poisoning in human beings, and especially its prevention."

The medical recommendations made by Dr. Duhig are as follow:

1. Constant inspection is necessary to detect small defects in routine work and machinery, and expert supervision constantly required to promote prevention of plumbism. This should be the duty of an expert of the Mines Department.

2. Atmospheric air analyses should be made in mine and mill immediately, and at least monthly at all those points suspected to be hazardous.

3. All high temperature processes, particularly welding and perhaps plumbing, should be carefully watched.

4. Prevention of plumbism and its reduction to a low minimum must be promoted by: (a) the employment of entirely fit men below middle age to start with; (b) elimination of susceptible persons by a probationary period of employment (four months is suggested); (c) periodical medical inspection of all personnel, as is done at present; (d) early treatment of mild cases detected in this way; (e) reemployment for a time in non-hazardous parts of the plant in special cases; (f) general welfare work, promotion of sport *et cetera*, and discouragement of the risk of alcoholism and venereal disease; (g) mechanical prevention of dust and fumes and the compulsory use of efficient respirators.

5. The diagnosis of plumbism for compensation purposes should be standardized. A form to this end should replace Form 7 at present in use is suggested.

6. The diagnosis of plumbism for compensation purposes should be solely confined to a board established for this end, to consist of a chairman and two other medical men appointed in terms of our joint recommendations.

7. Elimination from the industry of persons with a low lead tolerance must receive the sympathetic consideration of the proposed board.

8. The chairman of the proposed board should be required to conduct research into the physiological problems arising in the industry, and should be a person of high and special qualifications.

## British Medical Association News.

### SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Warrnambool on July 22, 1933. The afternoon programme consisted of a demonstration of clinical cases at the Warrnambool Hospital. Patients were presented by members of the honorary

medical staff and discussion took place afterwards on three selected cases. A report of this discussion will be published in a subsequent issue.

### Tubal Block and Sterility.

In the evening Dr. S. C. FITZPATRICK, of Hamilton, read a paper on "Tubal Block and Other Adnexal Lesions from the Aspect of Sterility" (see page 809).

PROFESSOR R. MARSHALL ALLAN said that he had been struck by Dr. Fitzpatrick's clear description of his cases and by the careful way in which he had presented his clinical results. Professor Allan emphasized the importance of the contraindications mentioned by Dr. Fitzpatrick. The operation of salpingostomy had been in vogue about 1910 in Dublin, but it had not been attended with much success. It should be noticed that Dr. Fitzpatrick's patients had suffered chiefly from block at the outer end of the tube. In cases of block near the isthmial and interstitial portions of the tube inflation had been fraught with less success. Rubens had pointed out the importance of early operation in cases diagnosed as appendicitis in young girls, because of the risk of subsequent adhesions with sterility or ectopic gestation in later years. Dr. Fitzpatrick's paper illustrated the importance of the method he used, even if it had led to success in only a few cases. Many workers in this field inflated the tubes again within twenty-four to forty-eight hours after operation and then at varying intervals, usually about once a month. Operations on women during the early months of pregnancy, with removal of the *corpus luteum*, almost invariably resulted in abortion. Professor Allan made light of his own small share in assisting Dr. Fitzpatrick by supplying references from the libraries. It was always a delight to him to be able to do this for men to whom the literature was not readily accessible.

DR. W. R. GRIFFITHS, referring to the piston-like action of the uterine dilator, said that while he was in Glasgow he had discussed this with the late David Shannon. Shannon, instead of dilating the cervix in cases of sterility, put the patient to bed, at complete rest, with daily hot vaginal douches for a fortnight, and then sent her home. This procedure was followed by as great a proportion of pregnancies as occurred after dilatation. With regard to the inflation of the Fallopian tubes, many workers thought that it was dangerous to use pressures greater than 200 millimetres of mercury, and some used even lower pressures than this. As a precaution against the occurrence of endometriosis, some workers recommended that inflation should be performed only five days after a menstrual period, as the repair of the endometrium was then complete and there was less likelihood of portions of the endometrium being forced into the peritoneal cavity.

DR. COLIN MACDONALD, appreciated Dr. Fitzpatrick's well balanced paper, though it was only the section on utero-salpingography that, as a radiologist, he was able to discuss.

In the Radiological Department of the Women's Hospital, Melbourne, a large number of lipiodol injections had been made into the utero-tubal cavities since the introduction of the method. Personally, he had found the interpretation of these utero-salpingograms not always easy, even when working in the closest cooperation with his gynecological colleagues. Like every other hollow muscular viscus, the Fallopian tube was lying in wait along the whole length of its course for the diagnostically unwary, with a well baited trap—the trap being that of spasm, and the bait being the apparent simplicity of diagnosis. At the uterine end of the tube was a physiological sphincter which might become spasmodic, often as the result of the intra-uterine injection; even a high intrauterine pressure might not overcome this spasm, with the result that an organic obstruction might be wrongly diagnosed. He had on several occasions seen a tube which appeared on one day to be obstructed either at the uterine end or further along its length, show next day complete patency. Dr. Macdonald knew of a case in which a left ostial obstruction was diagnosed, yet later a left tubal pregnancy developed. He therefore felt that before a diagnosis of obstruction was made from a utero-salpingogram it should



be confirmed on at least one after occasion. Some of the German authorities, recognizing this, advocated local anaesthesia by infiltration of the sacro-uterine ligaments to eliminate the misleading element of spasm. It was wrong to diagnose obstruction at the fibriated end until a film was made twenty-four hours after the injection. Often Dr. Macdonald had seen, in the film made immediately after injection, lipiodol held up at the fibriated end, whereas twenty-four hours later it was lying free in the peritoneal cavity.

Dr. Macdonald thought that Dr. Fitzpatrick might be interested in a film from the Women's Hospital collection. Immediately following the injection the patient, a Yugoslav who could speak no English, had a violent fit of coughing which promised seriously to interfere with the obtaining of a good film. The gynaecologist in charge, a fluent linguist, seemed to traverse the whole gamut of European languages, from Yiddish, the *lingua franca* of Eastern Europe, to the Spanish of old Granada, in an endeavour to arrest respiration, but without avail. However, all of a sudden the coughing ceased, and the film which Dr. Macdonald then showed was made. From the bizarre appearance the gynaecologist concluded that there was a communication between the uterine cavity and the bladder and that, by reverse peristalsis, the ureters had been filled with lipiodol. An interesting point had been that a film taken five minutes later showed practically no lipiodol remaining in the pelvis, though it had certainly not escaped into the vagina. The correct explanation of the film was that, owing either to excessive pressure or to unusual permeability of the uterine mucosa, the utero-ovarian venous and lymphatic systems had been injected with lipiodol; first the pampiniform plexus surrounding the uterus, and later the ovarian veins which ran up alongside the ureters.

This was the first such case that Dr. Macdonald had encountered, though a search of the literature showed that French and also Chinese observers were cognizant of the complication, which might have serious consequences, as lipiodol injected directly into the blood stream had produced death in laboratory animals. It was therefore unwise to use excessive pressure when injecting the oil, and fluoroscopic control, as in the pyeloscopic preliminary to pyelography, was desirable. At the Women's Hospital he made the injection about ten days after the cessation of the last period, at which time the permeability of the uterine mucosa was at a minimum.

In four cases of long-standing sterility Dr. Macdonald had seen pregnancy ensue shortly after lipiodol injection, and this therapeutic effect was probably due either to the overcoming of spasm or the separation of filamentous intratubal adhesions.

In reply, Dr. Fitzpatrick thanked the members for their appreciation. Referring to the pressures used in tubal inflation, he said that he had never used pressures over 200 millimetres of mercury, except on one occasion when he had used 250 millimetres with a successful result. Goodall, of Montreal, recommended taking the pressure up to 300 millimetres, and gave as his reason that on repeated inflation the pressure required to penetrate the occluded tube tended to be less on succeeding inflations, and Goodall considered that in cases presenting tubal obstruction no ill effect would result from pressures up to 300 millimetres. Dr. Macdonald rightly had emphasized the difficulty of interpreting lipiograms and the necessity for repeating them twenty-four hours later and even a week later. Catastrophes had occurred from dilatation of the cervix and they would occur unless the technique was correct and the contraindications were observed. The use of the method he had described was justified only in the hands of those who were prepared to give the time and effort necessary to perfecting the technique and the careful study necessary for the appreciation of the indications and contraindications.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held on October 26, 1933, in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. A. HOLMES A COURT, the President, in the chair.

### Lead Poisoning.

DR. G. C. WILLCOCKS read a paper entitled: "Lead Poisoning" (see page 813).

DR. CHARLES BADHAM read a paper entitled: "Basophilia and Lead Excretion in Lead Poisoning" (see page 816).

DR. R. J. MILLARD said that he was sure there were many others present who could discuss this subject better than he could. He thanked the readers for their very interesting papers. Dr. Willcocks, as the introducer of the subject, had dealt most succinctly with the clinical aspect. Dr. Badham had overwhelmed them with his data, theories and suggestions, which gave evidence of a deep knowledge of this important matter.

Dr. Willcocks had not referred to the advisability of disturbing this deposit of lead in the bones. The old idea was to give the patient iodides *et cetera* to get rid of it. Should they attempt to dislodge the lead or should they let well alone? If it was safely lodged in the bony tissues, could it do any harm there?

Dr. Millard had come across an interesting account of lead poisoning which had occurred at Baltimore in an unusual way. A poor class of negroes were using as fuel in their hovels the casings given them from a battery works. This went on for some months, when cases of lead poisoning began to occur. First one child was found with encephalitis, then another. When the authorities made an inquiry they discovered forty cases of lead poisoning which had arisen in this way. The battery casings carried a considerable deposit of lead salts. The fumes escaped into the ill-ventilated rooms and were inhaled, as was the soot. Both on analysis were found to contain lead. The brownish deposit on the rubber casings was mainly lead sulphate. No fatalities resulted from this outbreak. Of the forty patients there were five with encephalitis, six with nausea and vomiting, ten with headache and dizziness, thirty with a lead line on the gums; X ray examination showed a lead deposit at the ends of the long bones in twenty-five out of the forty cases. On examination of the blood, basophilia was found in twenty-three and anaemia in fourteen only. In some cases the symptoms were not clear. Interest was thus aroused and similar cases were discovered at Philadelphia, Detroit and Long Island. The exposure period for these forty cases was at least four months or more. It was not uncommon to get lead poisoning in people using painted wood for fires, though this was surprising in view of the comparatively small amount of paint that was burned in such a case.

DR. C. H. SHEARMAN thanked the readers for their interesting papers. Dr. Badham had mentioned that basophilic degeneration of the red cells was not seen in normal blood. While accepting this statement, Dr. Shearman emphasized the ease with which the condition could be missed. He (Dr. Shearman) had not realized this until he had commenced systematically examining blood smears for evidence of the condition. At least 20,000 cells must be examined in each film, and in each microscopic field the cells had to be studied almost individually to pick up basophilia.

The best stain appeared to be Giemsa. Leishman's stain was probably most frequently used in doing routine blood examinations, and unless great care was exercised to avoid overstaining, the conditions of basophilic degeneration would almost certainly be missed.

Dr. Shearman asked what number of cells showing basophilia per million Dr. Badham would regard as indicating lead poisoning. He (Dr. Shearman) and Dr. Shipton had been investigating at regular periods the blood of workers among lead in a factory near Sydney. The criterion that had been adopted by the head office of the company in America (on the recommendation of their medical officer there) was that a count of over 300 cells per million showing basophilia was to be regarded as indicative of plumbism and of removal of the individual to other work.

No cases of lead poisoning in this factory had occurred during the three years that these investigations had been carried out, and the highest number of cells showing basophilia found in any individual was 200 per million, though counts of 100 per million were not unusual. No notice

was taken of such counts because at subsequent examinations it was frequently found that these individuals would frequently show no basophilia.

Dr. Shearman would like to know whether Dr. Badham agreed that they were working along right lines in allowing these people with counts of 100 or 200 cells per million to continue working in lead.

With regard to basophilic degeneration, Dr. Shearman said it was a sign of blood regeneration and that immature cells were set free in the circulation.

In pernicious anemia, when regeneration occurred there was a relatively enormous increase in the reticulocytes of the circulating blood, but cells showing basophilia were not found in anything like the same proportion. A reticulocytosis of 10% (100,000 per million) was not uncommon, but counts of the same magnitude of cells showing basophilia were not seen. This was not because the condition was overlooked, as it would be practically impossible to miss the condition with such a high count.

In regeneration after anemia the cells showing basophilia in the circulating blood did not keep pace with the reticulocytes.

Dr. Shearman had understood Dr. Badham as quoting Davidson to have stated that in plumbism the reticulocytes and cells showing basophilia were equally increased, and Dr. Shearman would like to know whether this had been confirmed in Dr. Badham's investigations. If such were the case, it would appear that some condition occurred in lead poisoning which was absent in anemia to account for the relatively greater number of cells showing basophilia in the circulating blood during regeneration in the former condition. Could there perhaps be some change in the basophilic cytoplasm of the immature cell which was accentuated by the presence of lead so that the basophilic degeneration might have some analogy to the blue line seen on the gums?

DR. LORIMER DODS expressed his appreciation of the papers. He said that it was as important to be able to exclude lead poisoning in the diagnosis as to be able to identify it. He referred to the case of a girl aged five years who had suffered from headache, giddiness and vomiting. She had paresis of the external rectus muscle of the left eye and papilledema. There was only 0.01 milligramme per centum of lead in the urine. She was anemic, but no basophilia was found. Unfortunately, no X ray examination of the long bones was made. The child was seen by two senior physicians, who diagnosed a possible suprapituitary tumour. The family moved to Brisbane, where the child was seen by one of the Brisbane physicians, who promptly made a diagnosis of lead poisoning. The child was thus saved from possible optic atrophy and in all probability from having to undergo a serious surgical operation.

DR. LAWRENCE HUGHES said that the readers of the papers had raised many interesting points. He referred particularly to punctate basophilia, a condition which had interested him because of its association with malaria. He spoke of its occurrence during the convalescent period of blood regeneration. Dr. Hughes was interested to hear Dr. Badham say that punctate basophilia was indicative of lead poisoning. He (Dr. Hughes) had examined the blood of one hundred workers at a paint factory from the point of view of punctate basophilia. The condition had been present only in eight of that hundred, and then but moderately. There had been no count greater than 100. He wondered what the viewpoint should be.

Dr. Hughes had seen two patients this year with definite punctate basophilia, but whose blood picture was otherwise normal. He was at a loss as to what opinion to express in these circumstances.

Dr. Willcocks, in reply, said that there was only the first point, raised by Dr. Millard, for him to answer. That was in regard to the dislodging of lead deposits. He referred to the method recommended by Aub and Hunter, of administering calcium lactate and milk in order to fix the lead in the bones when the symptoms were acute. Later phosphoric acid or ammonium chloride could be used to dislodge the lead. But this was a dangerous procedure and should be carefully supervised. He himself had had experience only with iodides. The

procedure usually adopted appeared to be one of masterly inactivity.

Dr. Badham, in reply, referred to Dr. Shearman's finding of 300 stippled cells per million red cells in the blood of workers in a certain process and considered that what he called the group exposure to lead of this class of workers was quite small. If he could, as a routine, find anything as low as this in the hazardous processes in the battery works, he would be happy.

Yet even in workers at a process where the lead intake was small enough to produce only 300 basophile cells, Dr. Shearman might find workers who would complain of malaise and constipation, and a blood count and hæmoglobin estimation would reveal cases of mild lead poisoning. The presence of stippled cells in the blood was a very delicate test of the intake of lead. If there was doubt as to whether lead was being used or not in any process, it was easier to take blood smears from the people employed than to ask questions and receive untrustworthy answers, and the inquiry and chemical analysis of material could then be sharply focused.

Referring to the proportions of reticulocytes in the blood of individuals who were lead poisoned, Dr. Badham apologized to the Pathological Section for lack of work in this matter. He felt that they should in his division tackle this problem. There was much work to be done in this relationship of reticulocytes to punctate basophile cells. Though they had an excellent practical test for estimating the presence of lead and the severity of the workers' exposure to it, they were by no means happy in the chances they had to carry out more advanced research work.

In regard to what Dr. Hughes had said about punctate basophilia and malaria, he certainly agreed that in the tropics this matter would have to be specially investigated.

Referring to punctate basophilia and the diagnosis of lead poisoning, Dr. Badham said that they always checked this work by proving that there was an accompanying lead intake. The examination of the faeces led to fallacy, because the lead might not have been outside the liver. At one time a number of these faeces examinations were done, thinking that such an examination would be useful to show that the worker had purposely ingested lead. But such cases were not met with. Only once in the examination of 1,200 specimens was an attempt made to salt a specimen of urine—a teaspoonful of red lead was added to a Winchester bottle. The narrow limits of lead excretion made fraud possible only to a trained chemist.

DR. A. HOLMES & COURT, from the chair, said that they had been privileged to hear two papers of extraordinary interest and value. The small attendance, he assured them, was due to the inclemency of the weather and not to any lack of enthusiasm on the part of members. He was grateful to the readers of the papers.

#### NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Leake, David Norrie Fleming, M.B., B.S., 1932 (Univ. Sydney), Royal Hospital for Women, Paddington.  
Stabback, Reginald James, M.B., B.S., 1931 (Univ. Sydney), Lismore.

#### Correspondence.

##### THE HEALTH OF THE PEOPLE OF PAPUA.

SIR: There are about 300,000 natives in New Guinea, almost all of whom are infected with malaria and many with other diseases. The Australian officials will almost certainly become infected with malaria, and there will be, in all probability, a gradual development of a class of retired officers invalided and pensioned. The only real

remedy is the education of the indigenous population in the elements of medical science. With this end in view, the Government of the Commonwealth has made a commencement by sending a number of Papuans to the University of Sydney for six months' instruction.

So far as it represents initiative the action is praiseworthy, and it may appear at first sight somewhat ungracious to state that the policy is not by any means the best which could be pursued.

There is at Suva, Fiji, a native medical school at which selected men from seven groups of Pacific Islands are given a course of medical training of four years' duration, followed, if competent, by the issue of the Diploma of "Native Medical Practitioner". They then return to their native countries well trained and possessing that which few Europeans acquire, a knowledge of the language, customs and modes of thought of the people they live amongst. They become officers of health and are by general consent highly efficient. The cost is small and the only difficulty with regard to Papuans lies in the fact that for the admission of students to the school they must be able to read and write English thoroughly. Anyone who will read their medical journal, *The Native Practitioner*, will realize how capable they are technically, and how well they can express themselves.

All the European medical men who have visited the school speak in the highest terms of the work, and some of them have referred to the school as one of the finest pieces of work the British Government and the Rockefeller Foundation have accomplished in the Southern Pacific. The objection to the Commonwealth action is that Papuans are being given a wholly insufficient training and in a European atmosphere. There is really no comparison between the two methods, and criticism is based not on opinion, but on the facts of the case.

Without interfering with the Sydney venture, would it not be wise to send two or four selected Papuans to Suva and ascertain the result? They would be welcome in Fiji.

It is impossible, on economic grounds, to staff Papua with European practitioners, and it would be unwise to attempt it, even if large sums were available. If we are responsible for their welfare, it would be far better to let them take care of themselves, under European direction, after proper preparation, such as that provided at Suva.

Yours, etc.,

JAMES W. BARRETT.

103-105, Collins Street,  
Melbourne,  
November 21, 1933.

### Diary for the Month.

DEC. 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.

### Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser", pages xvi and xvii

ANTI-TUBERCULOSIS ASSOCIATION OF NEW SOUTH WALES, SYDNEY, NEW SOUTH WALES: Honorary Physician.  
BALMAIN AND DISTRICT HOSPITAL, SYDNEY, NEW SOUTH WALES: Medical Radiographer.  
DEPARTMENT OF INSPECTOR-GENERAL OF HOSPITALS, ADELAIDE, SOUTH AUSTRALIA: Resident Medical Officers.  
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer (male).  
MATER MISERICORDIE PUBLIC HOSPITAL, BRISBANE, QUEENSLAND: Honorary Assistant Relieving Surgeon.  
THE RACHEL FORSTER HOSPITAL FOR WOMEN AND CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Anaesthetists, Resident Medical Officer.  
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Resident Medical Officers.

### Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phoenix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

### Editorial Notices.

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